United States Court of Appeals

for the Second Circuit

APPENDIX

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Nos. 74-2284, 74-2286, 74-2308, 74-2450, 74-2449

JOINT APPENDIX TO THE BRIEFS

VOLUME VII

IN THE UNITED STATES COURT OF APPEALS
FOR THE SECOND CIRCUIT

THE SOCIETY OF THE PLASTICS INDUSTRY, INC.,

HOOKER CHEMICALS & PLASTICS CORP.,

UNION CARBIDE CORPORATION,

TENNECO CHEMICALS, INC.

and

AIR PRODUCTS AND CHEMICALS, INC.,

Petitioners,

v.

OCCUPATIONAL SAFETY AND HEALTH ADMINISTRATION,
UNITED STATES DEPARTMENT OF LABOR;
PETER J. BRENNAN, SECRETARY, DEPARTMENT OF LABOR;
and JOHN H. STENDER, ASSISTANT SECRETARY
FOR OCCUPATIONAL SAFETY AND HEALTH,

Respondents.

UNITED STATES COURT OF APPEALS
FILED
DEC 10 1974
A. DANIEL FUSARO, CLERK
SECOND CIRCUIT

On Petition For Review Of An Order Of
The Occupational Safety and Health Administration,
United States Department of Labor

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Products and Chemicals, Inc.

November 12, 1974

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2. Analysis by infra-red absorption

This is a potential alternative procedure for the direct analysis of air samples. There remains the question of detection limit or sensitivity. We have not yet had the opportunity to test the manufacturer's claims of being capable of meeting the very stringent requirements of the proposed standard. However, it is believed that the available instruments are capable of detecting vinyl chloride at a concentration of at least 10 ppm.

3. Direct analysis for vinyl chloride

Procedures such as gas chromatography and infra-red absorption are specific for vinyl chloride in air. Any procedure or instrument which does not provide for this specific analysis is considered completely unacceptable for the purpose of monitoring, since it provides unreliable or even misleading information. Included in this unacceptable category are those which determine only total hydrocarbons or organic compounds.

4. Direct analysis of the collected air samples without prior concentration or adsorption on inert materials

All concentration or adsorption steps should be avoided. They can lead to erroneous determinations due to losses of the volatile vinyl chloride during handling and introduction of contaminants from these operations, which may interfere with the accurate determination of vinyl chloride. Concentration and adsorption steps cannot be automated by presently available technology and therefore should be considered unsuitable for routine monitoring of regulated areas.

5. Collection of air samples in regulated areas in glass collecting tubes, stainless steel collecting tubes or plastic bags

Glass collecting tubes are recommended by NIOSH and have been found completely satisfactory in use under plant operating conditions. Stainless steel tubes are equally acceptable. The use of Tedlar or aluminized vinyl plastic bags has also been found completely satisfactory and reliable. Stainless steel tubing is recommended for the collection and conduction of air samples from a number of regulated areas to a central monitoring station. The use of hypodermic syringes to

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collect air samples for analysis is considered unacceptable since it does not provide for a positive seal of the sample from collection to the time of analysis.

6. Collection of personnel air samples in plastic bags

These have been found satisfactory, safe and reliable in plant use. Glass or steel collecting tubes are deemed potentially hazardous to employees and therefore, their use is not recommended. It is proposed that personnel air samples be taken over a relatively prolonged time period. For ceiling value determinations this time period should be in the 5 to 30 minute range, with 15 minutes serving as the preferable period. For TWA determinations a 6 to 7-1/2 hour period should be used.

Proposed Procedure for Obtaining Samples and Determining Vinyl Chloride Levels in Ambient Air

SCOPE:

This method is designed to sample and analyze for vinyl chloride in ambient air at concentrations between 1 ppm and 2% by volume.

PRINCIPLE:

Timed personnel monitoring samples are taken with small battery-powered pumps into aluminized vinyl bags. Vinyl chloride is determined directly in the air samples by gas chromatography using a flame-ionization detector (FID). A 10 ft. x 1/8 in. O.D. stainless steel column packed with the porous polymer Porapak S is used. The concentration of vinyl chloride is measured by comparing the peak height for vinyl chloride in the sample to the peak height in a synthetic mixture of vinyl chloride in nitrogen.

Environmental air samples are taken with pre-evacuated one-liter glass gas balloons. Opening of the Teflon valve will provide an almost instantaneous sample. Optionally the sampling time can be extended to 5 to 30 minutes by the insertion of approximate calibrated orifices into the sample inlet. The sample is then directly analyzed by the above procedure.

APPARATUS:

1. Hewlett-Packard 5750 Gas Chromatograph with flace ionization detector or equivalent.

2. Recorder with one millivolt span, one second full-scale response.

3. 10' x 1/8" stainless steel chromatographic column packed with 80/100 mesh Porapak S (Waters Assoc.).

4. Glenco gas-tight syringes 1 ml and 5 ml.

6. Calibrated Instruments portable battery-operated controlled flow pump(s). These pumps are available from the manufacturer preset for various pumping rates between 15 ml/min. and 223 ml/min. The rate can be changed by changing a single resistor on the control board of the pump. Further information is available from the manufacturer.

7. Calibrated Instruments aluminized vinyl sample bags, approximately 7 liter capacity.

8. Gum rubber tubing 1/8" I.D.

9. Vacuum pump.

REAGENTS:

1. Vinyl chloride, with a minimum purity of 95%.

2. Compressed nitrogen.

PROCEDURE:

A. Sampling System for Personnel Monitoring

1. Connect a portable pump with 15-ml/min. sampling rate to a 7-liter bag with a suitable length of gum rubber tubing.

2. Open the valve on the bag by turning counter-clockwise.

3. Clip the pump to the clothing of the person whose exposure is to be measured. The pump itself should be as close as possible to the person's nose and mouth as the sampling inlet is around the pump housing.

4. Fasten the bag to the person in a convenient manner, and switch the pump into the ON position. There will be a delay of approximately 30 seconds before the first sampling burst.

5. At the end of the sampling period, close the valve on the bag and turn off the pump. Samples should not be collected for more than 7 1/2 hours as the bags will burst from overfilling.

6. Disconnect the bag from the pump.

7. Evacuate a glass gas balloon to less than 0.5 mm Hg by connecting it to the vacuum pump.

8. Connect the bag to the evacuated balloon with the shortest possible connection.

9. Open the valve on the bag.

10. Open the valve on the gas balloon allowing the contents of the bag to flow into the evacuated gas balloon.

11. Close the valves on the gas balloon and the bag and disconnect the bag from the balloon. The gas sample is now ready to be analyzed by the gas chromatographic procedure below.

12. The bag can be emptied by pressing on the bag with the valve opened or by connecting the bag to the vacuum pump and thus evacuating the bag. The bag can then be reused.

B. Sampling System for Environmental Monitoring

1. Evacuate a glass gas balloon by connecting it to the vacuum pump.

2. Open the valve on the galss gas balloon allowing the air adjacent to the balloon inlet to be drawn into the collecting device.

3. The gas sample is now ready to be analyzed by the gas chromatographic procedure below.

C. Determination of Vinyl Chloride

1. Chromatographic conditions:

Injection port temperature: 150 °C
Detector temperature: 280 °C
Oven temperature: 115 °C
He carrier gas: 60 ml/min. at 75-80 psi
Air: 425 ml/min.
H_2: 40 ml/min.

Under these conditions vinyl chloride showed a retention time of 4 minutes.

2. Remove 1 ml of gas from the gas sample balloon with a gas-tight syringe and inject the gas into the chromatograph through the normal septum.

3. Replicate injections of a sample should produce peak heights for the vinyl chloride within 10% relative. Replicate injections may be desired until such reproducability of sample injection has been demonstrated. Ten 1-ml samples may be taken from one of the gas balloons without having a detectable effect on the contents of the balloon.

D. Calibration

1. For each calibration mixture, accurately determine the volume of a clean, glass balloon by filling with a suitable liquid and measuring the volume of the liquid. The balloons should be thoroughly dry before use for samples or calibration mixtures.

2. Flush a glass gas balloon thoroughly with vinyl chloride-free compressed air and close the valves.

3. In a thoroughly ventilated area, connect a suitable flow-controlling valve to a source of vinyl chloride and attach a length of latex rubber hose to the valve. Restrict the outlet of the hose to diminish back diffusion of air into the hose.

4. Open the valve and thoroughly flush the length of hose with vinyl chloride until free of air contamination.

5. With vinyl chloride flowing through the hose, insert the syringe needle through the wall of the hose.

6. Flush the syringe several times by withdrawing and depressing the plunger.

7. Withdraw the desired volume of vinyl chloride into the syringe. A good practice is to withdraw a slight excess of gas and then adjust to the correct volume immediately before injection.

8. Inject the vinyl chloride into the gas balloon through the side arm septum.

9. As both the balloon and the syringe were filled at atmospheric pressure, the concentration of any

calibration mixture can be calculated:

$$\text{vol \%} = 100 \times \frac{\text{Volume of vinyl chloride injected (ml)}}{\text{Volume of gas balloon (ml)}}$$

$$\text{ppm, volume} = 10^4 \times \text{vol \%}$$

10. To prepare calibration mixtures of less than 50 ppm, make dilutions by syringe from more concentrated mixtures. For example, to prepare a 10-ppm mixture, prepare a 1% mixture by adding 10 ml of vinyl chloride to a glass gas balloon filled with air. Remove 1 ml of the 1% mixture and inject into a glass gas balloon filled with nitrogen. The concentration is calculated:

$$\text{ppm by vol} = 10^6 \times \frac{\text{Volume vinyl chloride}}{\text{Volume Balloon I}} \times \frac{\text{Volume taken from Balloon I}}{\text{Volume Balloon II}}$$

E. Calculation of Vinyl Chloride Concentration

1. Chromatograph a calibration mixture under the conditions and technique given in Section B.

2. Choosing the calibration mixture resulting in a peak height for vinyl chloride closest to that observed in the sample, calculate the concentration in the sample:

$$\text{concentration} = \frac{\text{peak height sample} \times \text{concn. of mix}}{\text{peak height calibration mix}}$$

3. The concentration of the calibration mixture should always be of the same order of magnitude as that of the sample.

APPENDIX C

Proposed Alternative Standard for the Manufacture of Vinyl Chloride and Polyvinyl Chloride Resin Products Submitted by Stauffer Chemical Company

Comments by J. Wister Meigs, M.D.
Associate Clinical Professor, Occupational Medicine
Yale University

Stauffer Chemical Co. has proposed that exposures of workers to vinyl chloride (VC) vapor in the air of work places be limited to a 25 part per million (ppm) time-weighted average (TWA), with a 40 ppm ceiling maximum by October 5, 1974; that such exposures be reduced to 15 ppm TWA and 30 ppm ceiling maximum by October 5, 1975; and to 10 ppm TWA and 20 ppm ceiling maximum by October 5, 1976. They have asked me to comment on the health implications of this proposal.

The Stauffer proposal is a response to the proposed OSHA permanent standard of zero exposure to VC in the air, determined as less than 1 ppm ± 0.5 ppm. All these proposals recognize the occurrence of angiosarcoma of the liver in certain workers who had frequent exposures to high concentrations (several hundred ppm) of VC over periods of many years, generally at least 15 or more. Since January 1974, 20 cases of angiosarcoma of the liver have been identified throughout the world among present or former workers in VC plants, a population that may be in the 20,000 range. Probably less than 3,000 of these had exposures to VC for 15 years or more. It must be presumed that VC is itself a carcinogen. Animal exposures to chemically pure VC have been followed by cancer development. Mice appear to be more sensitive to this agent than other animals tested. Angiosarcomas

of the liver have been described in mice exposed to 50 ppm VC vapor 4 hr./day, 5 days/week for 2 1/2 years. Higher doses were reported to cause higher incidence of angiosarcoma.

The reasonable probability that VC can itself be a cause of angiosarcoma of the liver does not exclude the possibility that other chemicals or conditions in vinyl products manufacture may be important in angiosarcoma development, nor the probability that host factors and/or other environmental factors play contributory roles. Nevertheless, reduction of worker exposure to VC vapors should be followed, eventually, by reduction in incidence of angiosarcoma of the liver.

The presumption, in the proposed OSHA permanent standard, that, for health reasons, worker exposure to VC must be reduced at this time to zero poses both scientific and health policy problems, even if "zero" were to be redefined as 1 ppm. One difficulty lies in the implied assumption that the risk of angiosarcoma of the liver in vinyl products workers can be handled without reference to other environmental health risks. This implied assumption should be recognized as not only incorrect but possibly dangerous to health. For example, vinyl products manufacture entails risks of explosion. An engineering solution to employee exposure to VC that inadvertently increased explosive vapor risks might easily kill more employees from occupational accidents than it saved from angiosarcoma of the liver. Some years ago, an industrial hygiene engineering "improvement" in controlling a chemical that was explosive as well as toxic was the underlying cause of a later serious explosion.

Satisfactory control of health hazards to workers from VC demands the

broadest possible view. The word "satisfactory" is intended to imply that compromises must be made by all parties including the public. The example of possible explosion hazards of engineering "solutions" is only an indication of the complexities. There are many carcinogens in our environment. During the 20 years 1950-69 the male cancer death rate in the U.S. increased by almost 20%. Increases in lung cancer deaths accounted for more than half of this increase. Tobacco smoke, especially cigarette smoke, is clearly linked to lung cancer development. Perhaps it is a co-carcinogen with such materials as asbestos fiber or products of fossil fuel combustion, both generally and in specialized industrial exposures. Logical consistency would mandate an immediate and total ban on smoking materials of all kinds in all places of business coming under OSHA, if zero exposure to known carcinogens were to be adopted as the primary principle in dealing with them. Attractive as this might sound to those of us including myself who dislike tobacco smoke and avoid it whenever possible, such a ban might have consequences rivalling those of the Volstead Act in social undesirability.

OSHA has a responsibility to deal with this known and controllable carcinogen or co-carcinogen (tobacco smoke) in a reasonable way just as it should deal with vinyl chloride reasonably. For VC this requires consideration of responsibilities of other regulatory bodies in addition to OSHA, namely the Environmental Protection Agency and the Consumer Products Safety Commission. VC vapors are discharged from plants manufacturing vinyl products. Concentrations in the atmosphere around such plants have been estimated in the 1 ppm range, but definitive information is not yet available. Vinyl products themselves can be expected to give off small quantities of VC. Actual concentrations are believed, from theoretical calculations, to

be considerably less than 1 ppm, and a few measurements in support of this have been made, but further measurements need to be made under a variety of conditions of temperature, humidity, radiation (including infra-red and ultraviolet) and interaction with other chemicals in common consumer use.

Pending the availability of information as to actual and reasonably accurately estimated exposures of the general public to VC, around industrial plants using it, and, in consumer products, standards for exposures of workers to VC should continue on a temporary basis at the lowest levels known to be feasible within overall obligations to workers and the public. The obligations include the need to maintain jobs for employees and provide products to the public while questions of relative risk are being answered and protective steps are taken in reasonable and orderly sequence.

With respect to the proposed zero standard to be measured as less than 1 ppm, all evidence available to me suggests that it would be inappropriate at this time. Not only are there credible reports that it would be technically unattainable, but its acceptance would imply a degree of risk to the general public that isn't supported by current evidence. There may be better evidence on this question after cancer epidemiologists have studied time-trends and area experience in the incidence of angiosarcoma. From the Connecticut Tumor Registry we know that 2 cases of angiosarcoma of the liver were diagnosed from 1950-71, 2 more in 1972 and another 2 in 1973. Environmental histories of these patients are incomplete but exposures to plastics may have occurred in at least 2 of the recent cases. If one were to accept 2 excess cases of malignant disease per year in Connecticut as the current contribution of VC to the total Connecticut cancer burden, they would

represent less than 0.5% of the conservatively estimated annual contribution of tobacco smoke to Connecticut cancer cases and deaths in the form of lung cancer alone. Connecticut experience should be compared with that in other parts of the country. If evidence develops that angiosarcomas of the liver are beginning to occur in patterns indicating risks at extremely low levels of exposure, the plastics industry will need to devote effort and expense toward developing substitutes with less carcinogenic potential. In that event, heavy investment in reduction of employee exposure below levels suggested by Stauffer would seem misdirected. These more complex controls would operate for so short a time that their beneficial influence on cancer incidence would be marginal at best.

Evidence about angiosarcoma cases in populations whose approximate degree and length of exposure were estimated indicates a dose-response relationship. Cases occurred in subjects who had been exposed to several hundred ppm of VC for many years. No cases have been reported as yet in groups of men exposed for 10-25 years to VC concentrations in the less than 100 ppm range. A trend in one study toward excess numbers of metastatic cancers with primary site undetermined in men with heavier and longer exposure to VC raises the possibility that careful search will identify more cases of angiosarcoma of the liver as the primary site in subjects who die of metastatic cancer. If this possibility were to be confirmed, it would add urgency to the need for comprehensive evaluation of each option available for handling the overall risk. The decision as to the degrees and types of risk to which workers may be subjected will need review and up-dating at least annually until epidemiological studies of human experience have provided reasonably reliable estimates of relative risks of

exposures to measured and calculated past concentrations of VC either in manufacture or use of vinyl products. Animal studies will be needed to quantify and compare cancer risks from VC and other aspects of vinyl products manufacture or use with risks from all pertinent exposures that might occur in manufacture, distribution and use of substitutes for vinyl products.

The final decision whether vinyl plastics or other materials will be manufactured and sold must be reached with recognition of the fact that the public will eventually pay for the cost of the decision, and consumers should, therefore, be involved, through suitable representatives in the search for reasonable solutions to the problem. For the present, however, based upon the available medical evidence and human experience data, it is my opinion that the worker exposure levels proposed by Stauffer are reasonable.

J. Wister Meigs

J. Wister Meigs

New Haven, Connecticut
June 24, 1974

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Education & Training:

School-Sidwells' Friends School, Washington, D. C.	1920-32	Dipl.
College-Princeton University, Princeton, N.J.	1932-36	A.B.
Medical-Harvard Medical School, Boston, Mass.	1936-40	M.D.
Internship-Pennsylvania Hospital, Phila., Pa. (Rotating)	Nov. 1940-42	July
Residency-Mass. General Hospital, Boston, Mass. (Medicine)	Aug. 1942-43	Oct.

Medical Licensure: State of Washington, Department of Licenses, License to practice medicine and surgery, January 1945

State of Connecticut, Department of Health, License to practice medicine and surgery, September 1947

Membership:
American Board of Preventive Medicine (Occupational Medicine)
American Association for the Advancement of Science
American College of Preventive Medicine
American Medical Association
American Public Health Association (Fellow)
American Industrial Hygiene Association
American Conference of Governmental Industrial Hygienists
American Academy of Industrial Hygiene (Comprehensive Practice)
American Academy of Occupational Medicine (Fellow)
Connecticut Public Health Association
Connecticut Academy of Preventive Medicine - President 1966-67
Connecticut Industrial Medical Association - President 1967-68
Connecticut State Medical Society - Section on Preventive Medicine and Public Health - Chairman 1962-63
Industrial Medical Association
New Haven (City) Medical Association
New Haven County Medical Society
Sigma Xi

Employment History:

Alaska Division, Pan American Airways Seattle, Washington	Medical Officer, Oct. 1943 - August 1944

Employment: continued	Office of the Surgeon General United States Army	Major, MC,AUS: Chief Medical Section, October 1945 - July 1947, Army Industrial Hygiene Laboratory
	Yale University School of Medicine Department of Epidemiology and Public Health	Instructor, Assistant Professor, Associate Professor and Associate Clinical Professor of Occupational Medicine and Director of Preventive Medicine
	Yale University Department of University Health	Physician in charge of Employee Health Service
	U.S. Steel Corporation New Haven Works	Chief of Medical Services 1953-1968; Consultant Physician 1968 - present

Committees:

1. University Committees:

   Isotope Committee - 1960 - Present

   Safety Committee - 1967 - Present

   Trustee of Associates, Yale Medical Library (1967 - 70)

2. Outside of University:

   Committee on Occupational Health, Connecticut State Medical Society - 1948 to present, Chairman 1963-65

   Commission on Environmental Hygiene
   Armed Forces Epidemiological Board 1954 - 1972

   Member, Board of Directors, New Haven - Rehabilitation Center 1954, Vice President 1973

   Member, Medical Advisory Committee, Connecticut Industrial Nurses Association, 1958 - present

   Member, Health Planning and Coordinating Committee New Haven County Medical Society, 1968 - present

   New Haven Episcopal Deanery, Chairman, Grants Review Committee

   Fort Hunter Foundation (Fort Hunter Museum, Harrisburg, Pa.) President 1956 - present

Committees:
Outside University
continued

Member, Advisory Board, Fair Haven Health Clinic, 1972 - present

Director, Martin Luther King, Jr., Child Day Care Center, 1971 - present

Consultant on Safety and Health, United Illuminating Company, 1972 - present

Director, South Central Connecticut Regional Mental Health Planning Board, 1969 - present

Consultant on Occupational Health, Upjohn Company (and predecessor), 1949 - present

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J. Wister Meigs, M.D.

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EX 94a

TESTIMONY OF RICHARD J. HUGHES
Vice President of Union Carbide Corporation
Before
Department of Labor
Occupational Safety and Health
Administration

Vinyl Chloride Occupational Exposure Standard

My name is Richard J. Hughes. I am Union Carbide's Chemicals and Plastics Vice President, and I have served with the Company for over 25 years in this area. I intend to present the position of the Company, a New York Corporation with headquarters at 270 Park Avenue in New York City, with respect to the proposed OSHA standard on vinyl chloride, 29 CFR Part 1910, Section 1910.93g. We are and have been for 38 years a substantial manufacturer of polyvinyl chloride homopolymer and copolymer resins from vinyl chloride gas or monomer. Union Carbide helped develop the monomer in 1936, but we no longer make it ourselves. In other Corporate divisions, we are also a substantial user and fabricator of various products from the polyvinyl chloride resin.

Our interest in the development of a workable standard has coincided with the initiation of OSHA proposals for such action. We submitted a position paper on March 5, 1974 to the preliminary OSHA hearings on this matter held in Washington before Administrative Law Judge Gordon J. Myatt in which we stated that:

While we saw no need for a temporary emergency standard, where no emergency existed, we welcomed the initiation of the regular

rule-making procedure under Section 6 (b) of the Act, in which we are now participating, because it afforded a highly useful opportunity to investigate and review thoroughly and objectively the potential problems of vinyl chloride monomer handling p[illegible]es in industry. We were concerned that standards would be set without proper exploration of all phases of the matter. The institution and conduct of the present hearing accords with our stated objectives.

Our position in this situation is four-fold and may be summarized as follows:

1. There is no medical evidence provided by OSHA or NIOSH to justify the proposed OSHA standard of no detectable level of exposure, which evidence is required by statute. The evidence, set forth below, shows no need of such a restrictive limit.

2. We accept the temporary standard of 50 parts per million exposure level as safe. We support the SPI position as to lower levels of exposure. In fact, no tangible evidence has been presented at the present hearings that proves any lesser exposure is unsafe to humans.

3. We accept as a goal, a program to do whatever is technologically feasible to further reduce employee exposure to vinyl chloride.

4. We have an alternative proposal for OSHA, since we are sensitive to this problem and do not wish merely to be negative, as follows:

We recommend that OSHA proceed to establish a uniform regulatory work practice standard as suggested in draft language carefully prepared by Mr. R. N. Wheeler, Jr., and reviewed by many others. Some of the principles that appear therein have already been reflected in the proposed standard; others have not. We have already submitted it to OSHA on June 27, 1974 at this hearing, with the strong recommendation that it be carefully considered in the final rule-making. We have also submitted on the same date numerous comments on the proposed OSHA rule, which reinforces our view that it should be replaced with the standard we suggest.

We take this position for the simple reason that there has been no demonstration whatever that worker exposure to vinyl chloride monomer is hazardous at low levels of airborne concentration. Our own experience tends to document this contention. Little or no benefit or potential benefit to employees from over-stringent regulation has been shown, and industry statements and evidence on the economic impact of such action should cause concern over the continued ability of the producers to continue to operate. There is no legal warrant whatever, as the courts have already ruled, for establishing a standard at levels which no evidence supports. The OSHA statute permits court approval of a standard promulgated by the Department only if it is buttressed by substantial evidence on the record as a whole. Associated Industries v. Department of Labor, 487 Fed. 2d 342 (CCA2, 1973).

We do agree, of course, that our goal should be, wherever possible, to limit the exposure of employees by adoption of those engineering techniques and administration which are feasible. We agree that we should do whatever we can to install all technically feasible controls, and to continue to adopt safe and progressive work practices. Our concern, however, is that goals and legal requirements are two different things. A regulation should not presently require that which is achievable only in the future after the attainment of knowledge and technology that we do not yet have.

It should be emphatically stated that OSHA's proposed permanent standard cannot possibly be met through application of any known technology today, regardless of cost. In short, industry does not know how to comply with the recommendation. Adoption of the proposal as law would face the industry with the clear choice of either: (1) requiring its employees exposed to the extremely low levels of concentrations to wear clothing and respirators which are dangerous; cumbersome; uncomfortable; and resentment-building; which cause losses of employee productivity that will average up to 50 percent or more; and engaging in costly and time-consuming administrative monitoring, surveillance and record keeping; or (2) simply stopping production of the resin altogether with all the consequences that would ensue in the market place.

Indeed, it is not too much to say that the OSHA-proposed levels of exposure are unattainable, and unenforceable in practice

especially where non-continuous monitoring is adopted, even if such monitoring is faithfully carried out. One problem lies in the expected employee resistance to its effectuation, as a practical matter. It also would be simply impossible in many cases to ascertain whether a detectable level of vinyl chloride monomer has appeared in a given area, especially if, as occurs with most excursions, it would persist for only short periods of time.

Since no evidence that advanced engineering and modified production practices could ever achieve operations meeting a non-detectable level of vinyl chloride in the work-place air, the proposed standard will require, as a practical matter, all personnel in any restricted area to make 100 percent use of respirators at all times or for a major portion of the work day. Union Carbide holds that it is impractical and unsafe for employees to work regularly and routinely with respirators such as are required by the proposed standard. Considerable persuasive testimony has already been presented to this same effect at this hearing. The use of respirators should be considered only as a temporary practice when atmospheric levels of monomer rise above the permitted maximum and should be required only until corrective measures have been taken to bring the level of vinyl chloride down to or below the permitted maximum. As to respirator use, we submit that:

1. Due to current configuration of our plants, the fact that batch processing is employed, and the state of technology, the practical effect of the proposed permanent standard will be to require employees to wear respirators 100 percent of their time spent in the regulated area as described by the work standard.

2. It is difficult to imagine that workers in regulated areas would be willing to remain employed under these untenable conditions. Heat exposure; lack of mobility; severe limitations on communications with fellow workers; and the physical burden of respirator equipment will greatly lower employee morale over an extended period.

3. Workmen restricted by respirator equipment can precipitate safety problems due to awkwardness; limited ability to move to and from a unit due to emergency; restrictions on visibility; and fatigue from carrying and dragging cumbersome equipment.

4. Certain maintenance and operating tasks cannot be accomplished physically when a respirator is worn. The radius of activity is severely limited by length of hose, number and location of air supply outlets, and the weight and bulk of the respirator support apparatus. Moreover, the requirement of constantly checking supply source and hose to ensure operability is a burden.

Union Carbide has devoted much research, engineering, and production talent to bring its existing polyvinyl chloride

plants simply to meet the present standards and, thus, to minimize the time employees in the restricted areas need wear respirators, as required by the standard now in effect. We have uncovered no engineering approach to reduce vinyl chloride emissions to a non-detectable level. Relief from the standard that employees be required to wear respirators 100 percent of their time spent in the regulated area must come from the setting of practical levels of permitted monomer exposure.

The actual hazard, moreover, that is posed by any exposure of employees to vinyl chloride monomer is in fact a very limited one. It is always useful therefore to define what we are guarding against. Insufficient attention has to date been given to that simple fundamental; and the proposed standard itself avoids coming to grips with the matter. Instead, it attempts to settle the question by effectively forbidding all detectable exposure. Clearly this is "safe", but it hardly answers the problem. Indeed, it seems plainly to contravene the OSHA statute which abjures the Secretary to:

> "set the standard which most adequately assures, to the extent feasible, on the basis of the best available evidence that no employee will suffer material impairment of health or functional capacity." 29 USC Sec. 655 (b) (5).

The proposed standard hardly complies with that statutory command.

Yet there does appear to be a hazard to be met; and we agree with meeting it. What that hazard has been shown to be at this point is, that long-term exposure of employees on a continuous basis to vinyl chloride monomer emissions at high concentrations may cause liver angiosarcoma in some cases where inhalation takes place. The proposed standard is unnecessarily restrictive, technically and economically, in terms of such a limited hazard.

At this point, it would be appropriate to define areas where the proposed standard need not go at all. Thus, employees of resin fabricators, or any employee whose only potential contact with vinyl chloride monomer is to resins or products containing polyvinyl chloride resins whose retained monomer is less than .1 percent by weight, are exposed to no hazard and need no regulatory protection.

There is no need to be concerned about exposure to the polyvinyl chloride resin as such; it is chemically inert, not only as resin but also in the form of fabricated or mixed product. OSHA's nuisance dust standard with adequate ventilation adequately covers any exposure thereto. We need be concerned solely about the hazard said to result from exposure to the gaseous monomer, either during its own production; or during its reaction with other ingredients which produce the polymers; or retained in the resin and released into the atmosphere.

There has been, moreover, no showing that the monomer in its usual gaseous state causes any deleterious results by contact

with the skin, nor by ingestion. The retention of monomer on skin or clothing is unlikely because the product is volatile and non-adhesive, even where liquid monomer spills occur; a low boiling point assures such volatility. There could possible be some absorption through the skin, but the percentage is not known, nor has it been shown or claimed to have had adverse effects. A limitation will certainly pose no greater hazard to the skin than to the lungs.

So the hazard we need to meet is that of potential angiosarcoma after long-term inhalation exposure at high concentrations. Nothing else has been shown hazardous; any other hazard is simply speculation and nothing more, plainly not the sort of thing that would support a rule of law.

The proposed vinyl chloride monomer exposure standard of "no detectable level" in polymer operations implies that the health of workmen will be impaired materially if exposed even to traces of vinyl chloride. We know of no human or other medical data to support this conclusion. Actually, human experience in polyvinyl chloride plants operated by Union Carbide refutes it completely.

The solvent process (1) as operated by Union Carbide at Texas City has remained essentially unchanged as to process, recipe,

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(1) This is a process unique to Union Carbide, producing resins suitable for coating use.

equipment and operating procedure since about 1953. Its vinyl chloride emission are presently the same as has been experienced regularly over the past 20 years. Furthermore, measurements of vinyl chloride monomer in the workplace air and of exposure of operators to monomer made recently, are representative of the continuing condition since 1953.

Measurements during the first quarter of 1974 established that operators in the polymerization section of this plant were exposed to levels of vinyl chloride approximating 30 parts per million on an eight-hour time weighted average basis. Included in the tasks assigned to these operators is the taking and testing of autoclave samples to determine degree of conversion. During sampling and testing, which occurs four times each shift and requires about eight minutes to complete, workplace environmental levels can range as high as 400 parts per million in the sampling area. (We have taken steps to eliminate the need for this procedure).

Exposure time has been gathered on all of the 54 employees working in this job at the Texas City plant from 1948 to date. Twenty-four have from one to twelve years work experience on this job; two employees have had twelve years or more of exposure. No unusual liver function test results have been found based on a recent review of the medical evidence in the records.

In Union Carbide polyvinyl chloride suspension resin operations, the business realities since 1965 resulted in improved efficiencies in conversion of vinyl chloride monomer to polymer through modification of operating procedures and plant facilities. Increases in monomer efficiency (Polymer/Monomer charged x 100) from 85 percent in 1968 to 96 percent in 1974 at our Texas City plant have been and are being realized.. Improved machinery for recovering the monomer from vapors released in venting and stripping sections accounts for more than half of the improvement which resulted in significant reductions in monomer emitted into the workplace atmosphere.

Recent measurements within polyvinyl chloride suspension operations established that maximum vinyl chloride levels in the workplace air approximated 30 parts per million time weighted average (eight-hour) at a ceiling of 45 parts per million. Based on the known improvement in monomer efficiency, it is certain that the vinyl chloride monomer levels in the 1974 workplace environment are significantly below those of the prior years.

Here too, health records of employees at Texas City show no deterioration in the health of the workmen exposed during this period.

The Union Carbide experience is instructive in another respect. We operate several plants which fabricate a variety of

products from the polyvinyl chloride resin. Monitoring of our resin-handling at our fabrication plants show that the monomer levels (using a gas chromatograph sensitive to 0.15 parts per million) in the air around suspension resins rarely exceed 1 part per million at our calendars; 3-8 parts per million at our Banbury mixers; or an occasional "extreme" excursion to 18 parts per million in rare cases of exposure to the resin in the course of unloading. There has been discovered at those plants, moreover, after a check of employee medical records, not one case of angiosarcoma, nor any other liver or other disorder traceable to vinyl chloride. Many of our employees "exposed" to such concentrations are long-term employees who have worked on these substances for many years.

To return to the record at our Texas City plant, there have been 431 employees potentially exposed to vinyl chloride monomer at that location. This group includes all workers that could possibly have been so exposed. In it, there have been a total of 24 deaths since 1948. Not one has occurred from angiosarcoma. The exposure of these workers has, moreover, been lengthy and extensive. Seventy-four of them were exposed for more than 20 years. Ninety-seven were from 10 to 20 years. One-hundred ninety-one were exposed for 1 to 10 years, and 69 for less than one year.

Exposure levels at Texas City, furthermore, parallel those of many other older plants in Union Carbide and the entire industry.

The memory of many employees attests to the frequent detection of vinyl chloride odor over the years. Levels above the odor threshold were often reached in the early days. A recent test of levels of airborne concentration of vinyl chloride monomer which could be detected by odor was conducted by a panel of seven at our South Charleston, West Virginia plant. It was discovered that none of the panel could detect the monomer by odor at 133; 350; or 680 parts per million. Two of the seven detected it at 1200 parts per million. All seven detected it at 1960 and 3800 parts per million. The results support recent statements, including several made at the hearing, that vinyl chloride monomer levels in the past were far higher than thought initially. The limited dispersion of angiosarcoma in evidence found to date must be evaluated in the light of this knowledge.

Union Carbide has also produced polyvinyl chloride at our South Charleston, West Virginia, plant from 1936 to date. At one time, we also made the vinyl chloride monomer there; but discontinued such operations in 1968. After complete review of all available health records of a group of some 550 employees potentially exposed to vinyl chloride monomer at that location, where the early

exposure levels were far heavier than at Texas City, there have been discovered two cases of angiosarcoma. One employee, deceased in 1968, was exposed for 18 years, from 1944 to 1962, at high vinyl chloride concentrations. The other, still living, was exposed to high and medium high concentrations for approximately 24 years (1944 to 1968). The exposures were continuous over the years.

Yet many other employees plainly and obviously have been subject to the same high exposure levels and have suffered no ill effects therefrom. It is noteworthy moreover, that both reported cases --the only ones discovered out of a total of almost 1,000 Union Carbide polyvinyl producing employees reviewed-- were of individuals exposed in the earlier years of polyvinyl chloride production, when no concern about inhalation of the material was felt necessary. The only problem that arose then was at very high concentrations and was of flammability; employee protection was and is aimed at elimination of that hazard.

At one time, there was a body of medical opinion that felt that vinyl chloride monomer was useful as an ether substitute; other materials proved more adaptable as an anesthetic.

In operating polyvinyl chloride plants, we continue as in the past to give high priority to safe practices that will avoid the development of explosive concentrations of vinyl chloride monomer in the plant atmosphere and will eliminate ignition sources.

A defect in performance in this area of safety could lead to massive destruction of life and property. Thus, work practices must be written and carried out with avoidance of explosive concentrations and of ignition sources as the primary safety instruction regardless of exposure of workmen to monomer. As earlier indicated, such considerations did de-emphasize reliance on respirators as a safety device.

We therefore recommend that: (1) higher priority be given in the total safety procedure to isolating leaking pumps; sealing off ruptured lines; closing valves; blowing down autoclaves showing mechanical deficiencies; and taking all actions that will minimize the possibility of catastrophe from explosion and fire than to avoidance of exposure of workmen to even substantial concentrations of vinyl chloride for brief periods; (2) employees must be able to respond to upsets and emergency conditions not hampered by trailing airhoses or the 30-pound weight of a portable back-pack air supply; not immobilized by vapor-impervious clothing; and not cut off from vital communication by limited visibility and limited hearing; (3) it is sensible to accept a brief exposure to vinyl chloride monomer and thus to avoid a larger exposure or a greater hazard.

It is also noteworthy that concentrations of vinyl chloride monomer have been reduced over the years at the various American

producing plants, including our own. It is clearly a significant fact for these hearings that not one angiosarcoma case has occurred among the employees of a single one of the United States polyvinyl chloride producers whose operations began in or after 1950. It is also significant that, of the 13 United States cases so far reported, the average exposure of all such employees has been 19 years at high concentrations (i.e. above the odor threshold level) and that most of them had an exposure period whose onset antedates 1950. Moreover, the exposures that have resulted in the 13 reported cases occurred at only four of the 36 United States polyvinyl chloride producing plants. There are thus in the United States, 13 monomer producing plants and 32 polyvinyl chloride resin producing plants where no cases have been reported at all.

The conclusion is inescapable that the only hazard of potential angiosarcoma against which protection for employees is currently required is that of long-term exposure to high concentrations of vinyl chloride monomer. A level of exposure based on our recognition of the actual hazard would, we believe implicitly, pose no risk to the health of employees that could be in any way termed abnormal. Any extra limitations required would clearly be unnecessarily burdensome, and add nothing to improving worker health.

This is borne out in the recently issued Tabershaw-Cooper findings, released May 3, 1974, in which that organization reviewed

the records of a total of 8,384 polyvinyl chloride-exposed employees of whom at least 1,500 had been exposed for fifteen years or more, and all of whom had had at least one year of exposure prior to December 31, 1972. The findings were that the mortality rate of this group was only 75 percent of what would be expected in a comparable group of the United States male population; and that no cause of death in the polyvinyl chloride group studied showed a statistically significant increase over a comparable non-exposed group; yet all reported United States angiosarcoma deaths of vinyl chloride monomer exposees were found. The only caveat issued by Tabershaw-Cooper was that liver cancer occurred "more often than expected" in "those members of the group with the greatest exposure"; but that several other forms of cancer occurred at lower levels than those normally expected. However, the incidence of occurrence of other forms of cancer was not statistically significant.

A comparable study in Great Britain covering a group of polyvinyl chloride workers completed early in 1973, showed no increased incidence of cancer among the exposed workers, as compared to a control population; yet the British airborne concentrations of vinyl chloride monomer in 1973 were found to average 150 parts per million; from 1960 to 1970, to range from 300 to 400 parts per million; and from 1955 to 1960, 400 to 500 parts per million.

We also note with approval Dr. Dernehl's testimony reviewing an epidemiological survey whose conduct he oversaw which shows that liver abnormalities in the study group of employees exposed to vinyl chloride are comparable to those of employees in non-exposed control groups. Such a study of human reaction certainly does not suggest any need for an extraordinarily restrictive exposure level.

The only support, indeed, suggested for such restrictions has been studies not of humans but of rats and mice. The results of these studies, taken by themselves, are far from constituting conclusive or exhaustive proof of vinyl chloride monomer's carcinogenic effects at even the exposure levels set by the temporary standard currently in force.

Moreover, the transferability of mice studies to human experience is of dubious value. It is not known, indeed is doubtful, whether humans will react the same as mice to vinyl chloride monomer exposure. The only possible valid data that OSHA should consider in setting regulatory standards is that relating to humans. The wealth of such data confirms our belief that exposure at our recommended level is safe.

We are aware --and acutely so-- of the need of assuring every worker's health and safety. We have been in the forefront of such efforts for many years. Indeed, in 1973, our safety record was one of the best in the industry. Nevertheless, we see no merit

in OSHA establishing unnecessarily restrictive rules in the light of what we have defined above as the actual known hazard. Where there is no reason for regulation, it is folly to attempt it.

Summarizing our position once again:

1. There is no medical evidence provided by OSHA or NIOSH to justify the proposed OSHA standard of no detectable level of exposure, which evidence is required by statute. The evidence, based on human data, shows no need of any such restrictive limit.

2. We accept the temporary standard of 50 parts per million exposure level as safe. We support the SPI position as to lower levels of exposure. No evidence has been presented at these hearings to prove any lesser exposure is unsafe to humans.

3. We accept as a goal a program to do whatever is technologically and economically feasible to further reduce employee exposure to vinyl chloride.

4. Since we are sensitive to the problem and do not wish merely to be negative, we have submitted an alternative proposal for an OSHA standard.

There is merit in imposing limits on the vinyl chloride monomer and polyvinyl chloride manufacturers along the lines we have suggested. We believe that appropriate employee protection can result from their adoption.

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UNION CARBIDE CORPORATION
CHEMICALS AND PLASTICS
P.O. BOX 8004, SOUTH CHARLESTON, W. VA. 25303

SOUTH CHARLESTON PLANT

July 5, 1974

Mr. Julius Jimeno
Hearing Management Officer
Docket OSH-36
Room 200
Occupational Safety and Health Administration
United States Department of Labor
1226 M Street, N. W.
Washington, D. C. 20210

Dear Mr. Jimeno:

Attached are three copies of an additional appendix to be attached to the

TESTIMONY OF RICHARD J. HUGHES
Vice President of Union Carbide Corporation
Before
Department of Labor
Occupational Safety and Health Administration

Vinyl Chloride Occupational Exposure Standard

Very truly yours,

R. N. Wheeler Jr

R. N. Wheeler, Jr.

RNWJr/ra

Attachments

APPENDIX TO TESTIMONY
TO
RICHARD J. HUGHES
VICE PRESIDENT, UNION CARBIDE CORPORATION

A. PROPOSED STANDARD 1910.93q VINYL CHLORIDE
LEVEL OF EXPOSURE - METHOD OF ANALYSIS

B. PROPOSED STANDARD 1910.93q VINYL CHLORIDE
(m) TRANSPORTATION LOADING AND UNLOADING

C. VINYL CHLORIDE MONOMER THRESHOLD ODOR
CONCENTRATION IN AIR

D. VINYL CHLORIDE RESIN OPERATIONS

UNION CARBIDE CORPORATION
JOB CLASSIFICATIONS AND EXPOSURE
MONITORING

VINYL CHLORIDE MONOMER THRESHOLD ODOR CONCENTRATION IN AIR

The vinyl chloride monomer threshold odor concentration in air is an extremely critical value in attempting to evaluate employee vinyl chloride exposure in the early years of the polyvinyl chloride industry. Measurements of employee exposure to vinyl chloride were not made and, as a result, subjective judgments of employee exposure must be made based on whether the employee could smell vinyl chloride or not in the work place air. Based on a value for the threshold odor concentration of 260 ppm cited in the literature of the Manufacturing Chemists Association, the employee who could smell vinyl chloride in his workplace air was being exposed to more than .60 ppm.

The Union Carbide Corporation Plant Laboratory at South Charleston, West Virginia has investigated the threshold odor concentration of vinyl chloride monomer in air and has concluded that the threshold odor concentration is not 260 ppm but is approximately 2,000 ppm for a normal untrained person. This information indicates that the employee who smelled vinyl chloride in his workplace air was exposed to more than 2,000 ppm instead of more than 260 ppm as was previously believed.

The Laboratory report, showing the results of the Union Carbide Corporation study of vinyl chloride monomer threshold odor concentration in air, is attached.

RNWheelerJr/ra

July 5, 1974

Attachment

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JUL 05 1971
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PLANT LABORATORY

UNION CARBIDE CORPORATION
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VINYL CHLORIDE MONOMER THRESHOLD ODOR CONCENTRATION IN AIR

SUMMARY As the result of some discrepancies existing in the literature 1,2, for the Threshold Odor Level of vinyl chloride monomer in air, a study was initiated by the South Charleston Plant Odor Panel to determine the lowest detectable odor level of VCM in air. These data are tabulated in Table I.

DISCUSSION A series of six samples of vinyl chloride monomer and a blank were presented to the odor panel for determination of the Threshold Odor Level. The samples were prepared in increasing concentration over odorless distilled water in 250-ml Erlenmeyer flasks with stainless steel caps and submitted to a panel of seven people with instructions to record the first detectable odor. Each panelist tested the samples individually, so that there was no collusion among panelists.

A sample of the VCM vapor was then taken and analyzed by an AID Gas Chromatograph Model 511, hydrogen flame detector equipped with an 8 ft. column packed with Poropak Q 50-80 mesh. These data were corrected for the water vapor pressure and calculated to normal gas volume conditions, 0°C and 760 mm mercury. The tests and analytical data were duplicated.

CONCLUSION Individuals vary greatly as to the minimum concentration which is barely detectable. Our most sensitive odor panelists barely detected an odor of VCM at 1200 ppm. Normal odor detection level of VCM is at 2000 ppm. The normal detection level of VCM for the average untrained person is probably higher than 2000 ppm. An explanation as to why discrepancies have existed in the Threshold Odor Level is the fact that VCM is now a much purer commercial product than it was years ago and the odor detected was probably due to some impurity rather than VCM.

W. E. Griffith
Chairman
South Charleston Plant
Odor Panel

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TABLE I

THRESHOLD ODOR LEVEL OF VINYL CHLORIDE MONOMER IN AIR

Vinyl Chloride Monomer in Air ppm	Number of Panelists that Detected VCM Odor
Blank	0
133	0
350	0
680	0
1200	2
1960	7
3800	7

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Issued 1972 Page 6, Paragraph 3.1.2.
Manufacturing Chemists Association
Threshold Odor 260 ppm

2. Industrial Hygiene and Toxicology
Patty, F. A. and Associates
Interscience Publishers 1962
Volume 2 Page 1304
4100 ppm lowest level VCM odor can be detected.

3. Basic Principles of Sensory Evaluation
STP 433
American Society for Testing and Materials

4. Manual on Sensory Testing Methods
STP 434
American Society for Testing and Materials

5. Correlation of Subjective-Objective Methods
in the Study of Odors and Taste
STP 440
American Society for Testing and Materials

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VINYL CHLORIDE MONOMER THRESHOLD

ODOR CONCENTRATION IN AIR

The Odor Panelists have been screened for proficiency by the Arthur D. Little, Inc., Co. and have an average of 10 years experience in odor detections and evaluations. These screening test results are on file in the South Charleston Plant Laboratory.

Mr. Griffith, the chairman and moderator of the testing program is a graduate chemist and a member of the American Society for Testing and Materials E-18 Committee. This committee was founded in 1960 to promote knowledge, stimulate research, state principles and develop recommended practices in all areas of sensory evaluation of materials and products. Fields of interest currently represented include air pollution, clothing and textiles, metals and appliances, beverages and liquors, manufactured food products, flavors and fragrances, agricultural commodities, naval stores, tobacco, chemicals, paints and varnishes, paper and plastics, telephone, railroads, petroleum and allied products, laboratory equipment, packaging materials and pharmaceuticals. He is currently co-chairman of a Task Group to develop a recommended practice for odor of chemicals. Mr. Griffith represents the Kanawha Valley Union Carbide Locations on this committee.

M. A. Aitken
Director of Laboratories

MAA/JJh

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MEDICAL TESTING FOR LIVER CHANGES OF VINYL CHLORIDE WORKERS

Preliminary recommendations of
C. S. Davidson, M.D.
Massachusetts Institute of Technology

Routine Screening

The main purpose of routine screening is to pick up mild liver changes so that 1) workers can be moved away from exposure, 2) we can learn their frequency and distribution, and 3) prognosis and reversibility can be determined by long term follow-up.

I would suggest that all workers in a plant or a section of a plant where vinyl chloride is used (including management) be screened when this is logistically feasible. Clearly those with the greatest or longest exposure should be screened with high priority when, for any reason, all workers cannot be screened.

Screening procedures should include the following:

1. History--previous liver disease, jaundice, hepatitis, etc.; alcohol intake; usual diet; general health, fatigability, etc.
2. Physical examination--unlikely to produce much information except in advanced cases, yet I would recommend palpation of the abdomen for hepatosplenomegaly if for no other than psychological reasons.
3. Laboratory (not to the exclusion of other tests for general health usually done by the plant)
   a. Hematology--platelet count (by Coulter counter)
   b. Blood chemistry:
      1) total bilirubin
      2) both G & O transaminases
      3) alkaline phosphatase
      4) lactic dehydrogenase (and isoenzymes when elevated)
      5) gamma glutamyl transpeptidase
      6) gamma globulin (or serum protein electrophoresis)
   c. Other
      1) HBAg
      2) Carcino-embryonic Ag
      3) Alpha fetoprotein
      4) Ferritin

4. Follow-up--If any of the above are positive, the tests should be rerun. If then negative, rerun again in approximately three months. If one or more tests is persistently positive, the worker should be removed from any exposure, investigated by a physician further, and at least reevaluated again in three months. If tests are strongly or persistently positive, liver scan should be done for splenomegaly. If the scan suggests a liver lesion an arteriogram or biopsy should be considered, but now we are looking for tumor with the hope of cure, i.e. medical care. Whether biopsy, laparoscopy or laparotomy should be done is a "medical" decision upon which one cannot lay firm recommendations at present.

All workers with negative history, physical examination and laboratory tests should be reexamined every six months. It may well be that workers with minimal exposure (eg. secretaries) might eventually be shown not to need these repeated screens, but I suggest that they be done on all at present to control for minimal exposure and for reassurance.

B. Research

1. Vinyl chloride effects on animals (lower and non-human primates) should be studied intensively for short and long-term effects on the liver (as well as on other tissues).
2. Attempts should be made to immunize animals with angiosarcoma in the hope of having antisera capable of picking up these sera.
3. Sera from patients with angiosarcoma and with persistent liver abnormalities and without should be tested for the presence of "F antigen". This is a new, liver specific antigen which is occasionally positive in other liver diseases, although not yet, in our hands, in some 30 instances of hepatocellular carcinoma or one case of angiosarcoma in a child. This is highly preliminary work.
4. As many as possible of the workers who have persistently positive tests should be studied in clinical research centers where in-depth studies can be made (with informed consent,

of course).

Search should be made in blood for changes in the "micro-proteins" many of which are made in the liver. If Dr. Popper's hypothesis of early collagen formation in the sinusoids is to be studied, collagen enzymes and fragments should be looked for.

The presence of what may be many macrophages between the liver cells and the sarcoma cells suggests that immunologic study of both sarcoma patients and presarcoma patients (if any) be studied with modern methods for both humoral and cellular immunity.

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UNIVERSITY OF SOUTHERN CALIFORNIA
SCHOOL OF MEDICINE
DEPARTMENT OF MEDICINE
LOS ANGELES, CALIFORNIA

REPLY TO
[illegible] HOSPITAL
2069 SOUTH HOPE STREET
LOS ANGELES, CALIFORNIA 90007

TELEPHONE
AREA CODE 213
747-5211
EXT. 370

March 7, 1974

Clark W. Heath Hr, MD - Chief
Cancer & Birth Defects Division
Building 1, Room 520
Center for Disease Control
ATLANTA, GA 30333

Special Delivery

SUBJECT: Review and Recommendations concerning Hepatic Angiosarcoma apparently related to vinyl chloride exposure (Conference of 3/2/1974)

Dear Dr. Heath:

At the conference at the NIH on March 2, 1974, data was presented concerning 6 proven cases of angiosarcoma of the liver (possibly a seventh case) occurring at the B.F. Goodrich plant at Louisville, Ky. On the basis of the exposure there to vinyl chloride, the cases presented by Dr. Popper from Germany and the animal studies mentioned, it seems essentially inescapable that the lesions occur incident to the exposure to vinyl chloride. There were a number of similarities among these cases. The tumor pathology was identical and the tumor behavior seemed to be one of local extension rather than widely metastatic. Case #1 might be tumor of multicentric origin.

The liver lesion in the non-tumor portions of the liver was was of great interest. Material from four cases without known tumor was reviewed, presenting similar pathology. Primarily, there was widening and fibrosis of the portal areas, some collagen in the sinusoid near the periphery of the lobule and peculiar sinusoidal lining cells, presumably the precursors of the angiosarcoma cells. It would appear that the portal fibrosis, or, maybe, more

2/

importantly, the sinusoidal fibrosis leads to fairly early portal hypertension, hemodynamically similar to idiopathic portal hypertension (Banti's disease). Esophageal varices and marked splenomegaly were present in some of the cases.

Unfortunately, neither the tumor nor the liver lesion preceding it provoke very marked alterations in the hepatic tests. The most consistent appears to be small to moderate increases in the SGOT, some increase in serum alkaline phosphatase and sometimes small rises in the serum total bilirubin. Other tests seemed to be variably abnormal (LDH, serum globulin). Coupled with the mild test abnormalities was increased spleen size in a number of the cases.

The central problem is that of how to monitor exposed workers for early evidence of hepatic toxicity. It would appear that the hepatic lesion precedes tumor formation, as a rule, by some years. Also that there is some relationship to a long duration of exposure for both lesions. It is recognized that rather large numbers of workers are involved for screening purposes. My suggestions are as follows:

1. Biochemical screening at the time of new employment (base line) and for all workers, then once per year. I would suggest this be increased to every six months after the 10th year of exposure.

2. Preferably the biochemical tests should be those able to be done by the SMA (or other automated means).

3. It might be of use - experience would tell - to program the SMA for the SGPT (as well as SGOT) and possibly for the GGTP. This type of liver lesion appears to produce little hepatocyte injury and thus transaminase-type tests are going to be of limited value, but there may be nothing currently better.

4. More intensive investigation could then be done on the cases that drop out with test abnormalities. I would suggest repeat tests in one month, possibly again in another month. Hopefully the abnormalities will be modest in number and therefore practically could be investigated more thoroughly.

The hepatitis B antigen probably should be done when the enzyme tests are elevated to help clear the picture of unrelated viral hepatitis. If abnormalities persist one month, I would advise then a physical examination, history taking (alcohol, medications, recent transfusion or hepatitis contacts, etc.)

¶5. If the cause of the hepatic test abnormalities is not clear, i.e. now possibly vinyl chloride-related liver disease, a liver scan with technicium would seem indicated and useful. As discussed at the conference, at this point practicably it is likely the patients would be referred to private physicians or clinics for further work-up.

¶6. There are a number of other considerations. The proline hydroxylase test might be of interest and utility in screening. The BSP test seems not to be (not surprising, in light of the Banti-like disorder). It remains uncertain if the hepatic lesion might still advance to tumor with withdrawal from vinyl chloride exposure. I doubt this tumor will be associated with alpha-fetoprotein, but this should be determined, especially by radioimmunoassay.

¶7. I doubt that unusual biochemical tests will turn out to be of much value over customary hepatic tests, since the hepatic tests are usually based on reduction of functional mass, hepatocyte injury or bile duct obstruction, none being prominent here. Nonetheless some rather intensive investigation of a tumor case and a few of the "liver disease" cases might provide useful information applicable to screening.

¶8. Liver scan features showing some degree of splenomegaly or "spotty" hepatic uptake probably should be arbitrarily interpreted in this setting as vinyl chloride toxicity.

¶9. Liver biopsy, when indicated as considered by physicians caring for these patients, can be very helpful, it appears, and should be encouraged. However, if the scan suggests tumor type of defects, needle biopsy might be somewhat hazardous with this vascular type of tumor.

10. The idea of monitoring a "control" population was raised and is attractive if feasible.

11. An effort should be made to follow up former exposed employees to see what has occurred after leaving exposure areas.

12. For workers in high exposure work (autoclaves), some of the techniques used in the lead industry (air currents, gas masks, showers, etc.) might be of value in reducing the degree of exposure.

13. There seems to be urgent need for data re the biology of vinyl chloride (metabolism, excretion, tissue levels, etc.)

Very truly yours,

Allan G. Redeker

Allan G. Redeker, MD
Prof. of Medicine
Director, Liver Unit,
John Wesley Hospital

MD

Jah

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The B. F. Goodrich Company

500 SOUTH MAIN STREET • AKRON, OHIO 44318 • PHONE: 216-379-2126

ADDRESS REPLY TO: DEPT. 0001 BLDG. WHB-4

DIRECTOR OF ENVIRONMENTAL AFFAIRS

July 12, 1974

Mr. Julius Jimeno
Docket 36
1726 M Street, N.W., Room 200
Washington, D. C. 20210

Dear Mr. Jimeno:

I am enclosing our laboratory report which fulfills the request by the Office of the Solicitor for further information on the technical details of testing commercially available charcoal filled canister and cartridge protective breathing devices.

The experimental approach is described in detail. The wide range in capacity for absorbing vinyl chloride by commercial absorbants is shown in Table I on page 4 and the clear superiority of activated charcoal is evident. The comparison of cartridge and canister data in Tables 2 and 3 indicates that depth of carbon bed is also a factor in capacity which must be balanced against pressure drop in the design of a respirator for breathing protection. The desorption experiments show that the recharging of cartridges and canisters is feasible.

Laboratory work is continuing. If further pertinent information develops, it will be submitted before August 23, 1974.

Very truly yours,

THE B.F.GOODRICH COMPANY

Roger W. Strassburg

Roger W. Strassburg, Ph.D.
Director of Environmental Affairs

k

Enc.

TO		
Dr. R. W. Strassburg	Akron; Loc. WHB-4; Dept. 600	
FROM		DATE THIS LETTER IS
Dr. M. M. O'Mara	Avon Lake Technical Center	July 2, 1973

SUBJECT

An Investigation of the Adsorption Properties of Vinyl Chloride Monomer on Commercial Carbon Filters

Introduction

This report is a summary of our work on the adsorption characteristics of VCM on carbon filters which are used in conjunction with protective breathing masks. The basic objective of the study was to determine the adsorptive and desorptive characteristics of various, commercially available, carbon filters which are used as protection against inhaled organic vapors. This study involved vinyl chloride monomer only.

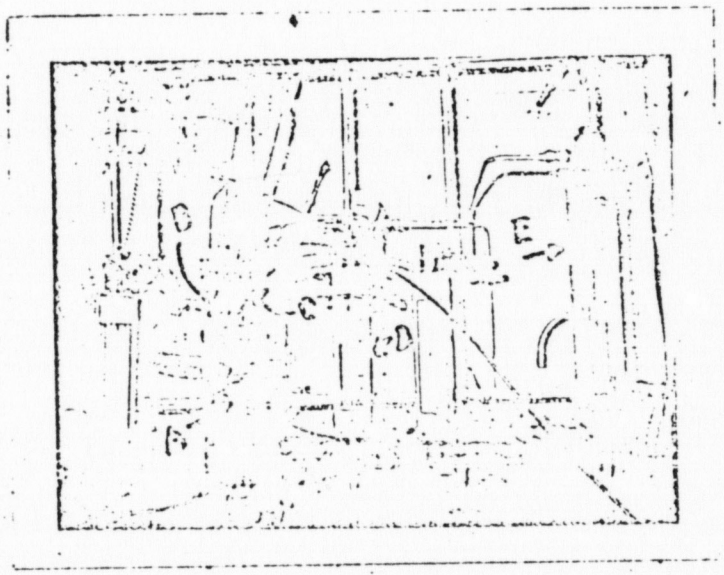
Experimental Approach

An experimental flow chamber was constructed so that carbon filled cartridges and canisters could be subjected to a continuous flow of air containing vinyl chloride monomer. A continuous rather than batch monitoring method of analysis was chosen to indicate the presence of vinyl chloride monomer. A photograph of the basic flow chamber is shown in Figure 1. The vinyl chloride-air stream enters the carbon device at A. The scrubbed stream then exits the device at B. Line C is the continuous monitoring system for sensing vinyl chloride monomer in the air stream. Additional experimental data are contained in the Appendix. To demonstrate the type of data that is obtained from the experiment described above, a plot of VCM concentration versus time is shown in Figure 2. The rise in the horizontal baseline indicates incipient vinyl chloride monomer breakthrough.

In our initial experimental design, we used a portable Century Organic Vapor Analyzer to sense vinyl chloride monomer. The major problem with this analyzer was its inherent background which could not be subtracted electronically from the recorder output. To overcome this we replaced this instrument with a Bendix Hydrocarbon Analyzer. Both instruments are flame ionization units; Figure 2 is based on the output of the Bendix instrument. The Bendix instrument is well suited for the type of measurement carried out because of the following unique characteristics:

(1) low drift
(2) linear (vs. logarithmic) amplifier
(3) background zero suppression
(4) excellent stability

Figure 1: Experimental VCM Flow Chamber



(A) vinyl chloride/air stream into cartridge holder

(B) cleaned air stream exit

(C) analyzer sampling line

(D) vinyl chloride flowmeter

(E) high capacity air flowmeter

Figure 2

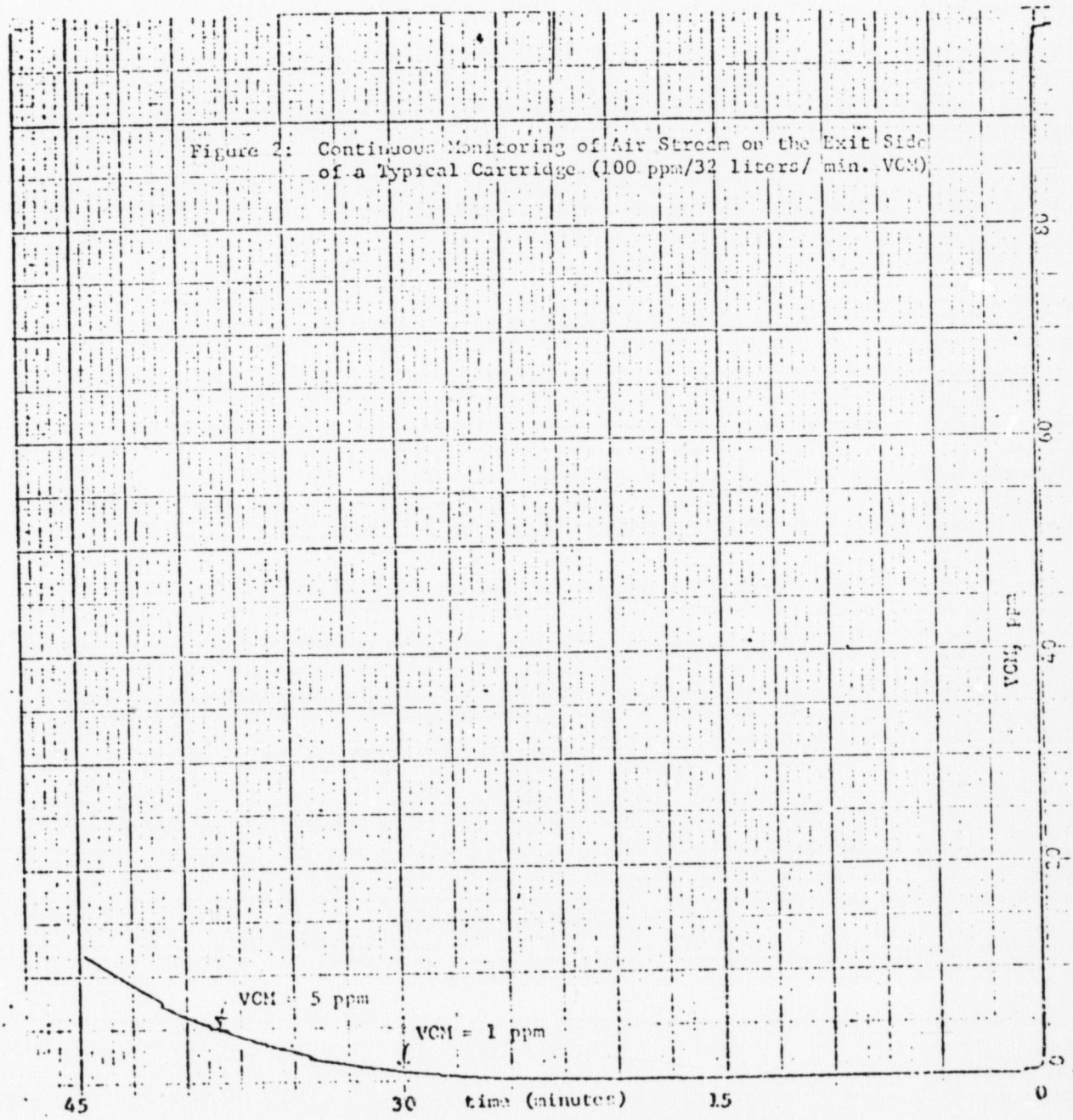


Figure 2: Continuous Monitoring of Air Stream on the Exit Side of a Typical Cartridge (100 ppm/32 liters/ min. VCM)

Experimental Results

(1) Initial Evaluation of Commercial Cartridges

A variety of commercial cartridges were evaluated with the Century analyzer in order to obtain a profile of their ability to retain vinyl chloride monomer. All evaluations were carried out at a flow of 30 liters per minute and a vinyl chloride concentration (volume/volume) of 100 parts-per-million. In these early evaluations, the time to 5 parts per million of vinyl chloride at the exit side of the carbon matrix was determined. This value is referred to as T_5 (in minutes). Table 1 contains a summary of the various cartridges evaluated and the corresponding T_5 values obtained.

Table 1

T_5[1] Values for Various Carbon Cartridges
When Tested at 30 liters/minutes and 100 ppm of Vinyl Chloride Monomer

Cartridge	T_5[1], minutes
Welsh 7500-1	41, 34, 39, 44, 33
American Optical AOR55	21
Westvaco Nuchar WV-H 8X30	15
Willson R-25	34
Willson R-11	0
American Optical R-51	29
Willson R-21	42 ± 13
Welsh 7400-1L	93
Welsh (Canister) 7800-1M	678
MSA Cartridge 44135	13

[1] T_5 refers to the time in minutes for the concentration of VCM at the exit end of the cartridge to reach 5 ppm.

It can be seen from these data that the commercial cartridges and canisters chosen for this study exhibit a broad range in ability to absorb vinyl chloride monomer. All of the 3-inch cartridges were effective from zero minutes (no VCM adsorption) to about 40 minutes. After initial vinyl chloride breakthrough there is a gradual rise to a value corresponding to the concentration of vinyl chloride in the air stream.

As expected, the 4-inch cartridge (Welsh 7400-1L) and the canister outperformed the 3-inch cartridges under these test conditions. It should be noted that in the end-use mask construction, two of the 3-inch cartridges are used. On the other hand, a single 4-inch cartridge or canister is used in masks employing these carbon devices. Thus to obtain a true evaluation of the 4-inch cartridge and canister, they must be exposed to an air flow that is double that used for the 3-inch cartridge evaluation. This type of evaluation is described below.

(ii) Detailed Evaluations of the Welsh Canister, 3-inch Cartridge and 4-inch Cartridge

The NIOSH requirement for testing protective masks calls for evaluations at the following atmospheric flow rates: 32 liters/minute and 64 liters/minute. The Welsh canister (7800-1M), the Welsh 4-inch cartridge (7400-1L) and the Welsh 3-inch cartridge (7400-1) were evaluated within these guidelines. Testing with the 3-inch cartridge was restricted to a flow rate of 32 liters/minute while that for the 4-inch cartridge and canister was limited to a flow rate of 64 liters/minute. In order to improve the accuracy of the measurement, a Bendix Total Hydrocarbon Analyzer was obtained for monitoring vinyl chloride monomer. Using this instrument in the experiment approach described above, measurements at 1 ppm VCM breakthrough (T_1) could be accurately obtained. Table 2 contains a summary of T_1 values obtained for the three Welsh cartridges and canisters. These data show that vinyl chloride breakthrough (T_1) is essentially the same for the 3-inch and 4-inch cartridges when tested at the two required flow rates. It is also obvious that breakthrough is quite dependent upon concentration. For the two cartridges, a 4-fold increase in VCM concentration caused a 50% increase in breakthrough time. The stronger dependence of VCM/air rate on breakthrough time is demonstrated by comparing data in Table 1 with that in Table 2. For example, comparing T_5 data (accurate T_1 data is not available in Table 1) it is seen that for the 4-inch cartridge there is a 50% increase in breakthrough (T_5) when the rate is increased from 30 liters/minute (T_5 = 93 minutes) to 64 liters/minute (T_5 = 48 minutes) at a VCM concentration of 100 ppm.

Table 2

T_1 [1] Values for the Welsh 3-inch, 4-inch Cartridges and Canister at Various Concentrations of Vinyl Chloride Monomer

I. Welsh 7500-1 (3-inch)

32 liters/minute

VCM, ppm	T_1, minutes
25	80
50	60
100	35 (T_5 = 44 minutes)

II. Welsh 7400-1L (4-inch)

64 liters/minute

VCM, ppm	T_1, minutes
25	70
50	60
100	40 (T_5 = 48 minutes)

III. Welsh 7800-1M (Canister)

64 liters/minute

VCM, ppm	T_1, minutes
25	522 (8.7 hrs.)
100	342 (5.7 hrs.)
1000	102 (1.7 hrs.)

(1) T_1 refers to the time in minutes for VCM concentration to reach 1 ppm.

Considering only those data in Table 2 it can be shown that the amount of vinyl chloride adsorbed increases with increasing concentration of vinyl chloride in the air stream. The absolute amount of vinyl chloride adsorbed by the carbon in each of the experiments can be obtained from the following equation:

$$\text{VCM, cc} = \text{total rate (cc/min.)} \times \text{concentration VCM}\left(\frac{\text{ppm}}{10^6}\right) \times T_o$$

where T_o is initial breakthrough at "zero" parts per million.

Once obtained, relative VCM adsorption efficiences can be calculated by dividing these values by the weight of the carbon in the cartridge. A summary of these data are contained in Table 3. It is evident that the efficiency of carbon to absorb vinyl chloride monomer is not constant but rather increases as the concentration of vinyl chloride monomer increases in the air. It is significant to note that the efficiency of carbon to adsorb vinyl chloride monomer is nearly constant for the two cartridges when compared at the same vinyl chloride concentration, even though the flow rates are significantly different. This sameness in efficiency, of course, relates back to the previous statement that these two cartridges offer the same degree of protection. As a final comment the very high efficiency of the carbon bed in the canister is responsible for the excellent (relative) protection this device provides.

It appears that emperical mathematical equations describing breakthrough times for the two types of cartridges could be readily developed since the adsorption efficiency at a given VCM concentration is constant. However, before this is attempted more data would be necessary at different flow rates and over a broader concentration range.

Table 3

Calculation of Absolute Adsorption of VCM (cc)
on Carbon and Efficiencies (cc VCM/g Carbon)

I. Welsh 7500-1 (3-inch)

32 liters/minute

VCM, ppm	VCM Adsorbed, cc	Efficiency(1), cc/g
25	48.0	1.34
50	82.4	2.29
100	108.8	3.03

II. Welsh 7600-1L (4-inch)

64 liters/minute

VCM, ppm	VCM Adsorbed, cc	Efficiency(2), cc/g
25	88.0	1.31
50	160.0	2.38
100	227.0	3.39

III. Welsh 7800-1M (Canister)

64 liters/minute

VCM, ppm	VCM Adsorbed, cc	Efficiency(3), cc/g
25	777.6	2.51
100	1958.4	6.31
1000	6336	20.4

(1) cartridge contained 35.9g carbon

(2) cartridge contained 67.0g carbon

(3) canister contained 310.3g carbon

(iii) Desorption of Vinyl Chloride Monomer

It was of intevest to study the desorption characteristics of vinyl chloride monomer from the cartridges. To accomplish this objective, the 3-inch Welsh cartridge was exposed to vinyl chloride monomer at 100 ppm and 32 liters/minute until initial breakthrough at 1 ppm (i.e. T_1) was achieved. At this point the vinyl chloride feed stream was shut off and the cartridge was exposed to clean air at 32 liters/minute. A skewed bell-shaped curve developed which maximized at 33 ppm (time = 84 minutes) and decreased to 0 ppm (time = 330 minutes). In actuality we were producing a gas chromatogram in which the carbon canister was a crude chromatographic column, the air was the carrier gas, the vinyl chloride was the sample and the Bendix analyzer was the detector. It is well known that one way to decrease the retention time in gas chromatography is to increase the flow rate. To demonstrate this phenomenon with the present system, we repeated the above experiment (3-inch cartridge, 100 ppm VCM, 32 liters/minute) except once initial VCM breakthrough occurred the vinyl chloride feed was discontinued and the clean air rate was increased to 64 liters/minute. At this increased flow rate, the bell shaped "chromatographic" curve was less skewed. The maximum now occurred at 36 ppm (time = 49 minutes) and decreased to 0 ppm (time = 194 minutes). This trace is shown in Figure 3. Thus by doubling the "carrier gas" flow rate from 32 liters/minute to 64 liters/minute, the "retention time" decreased by 42%.

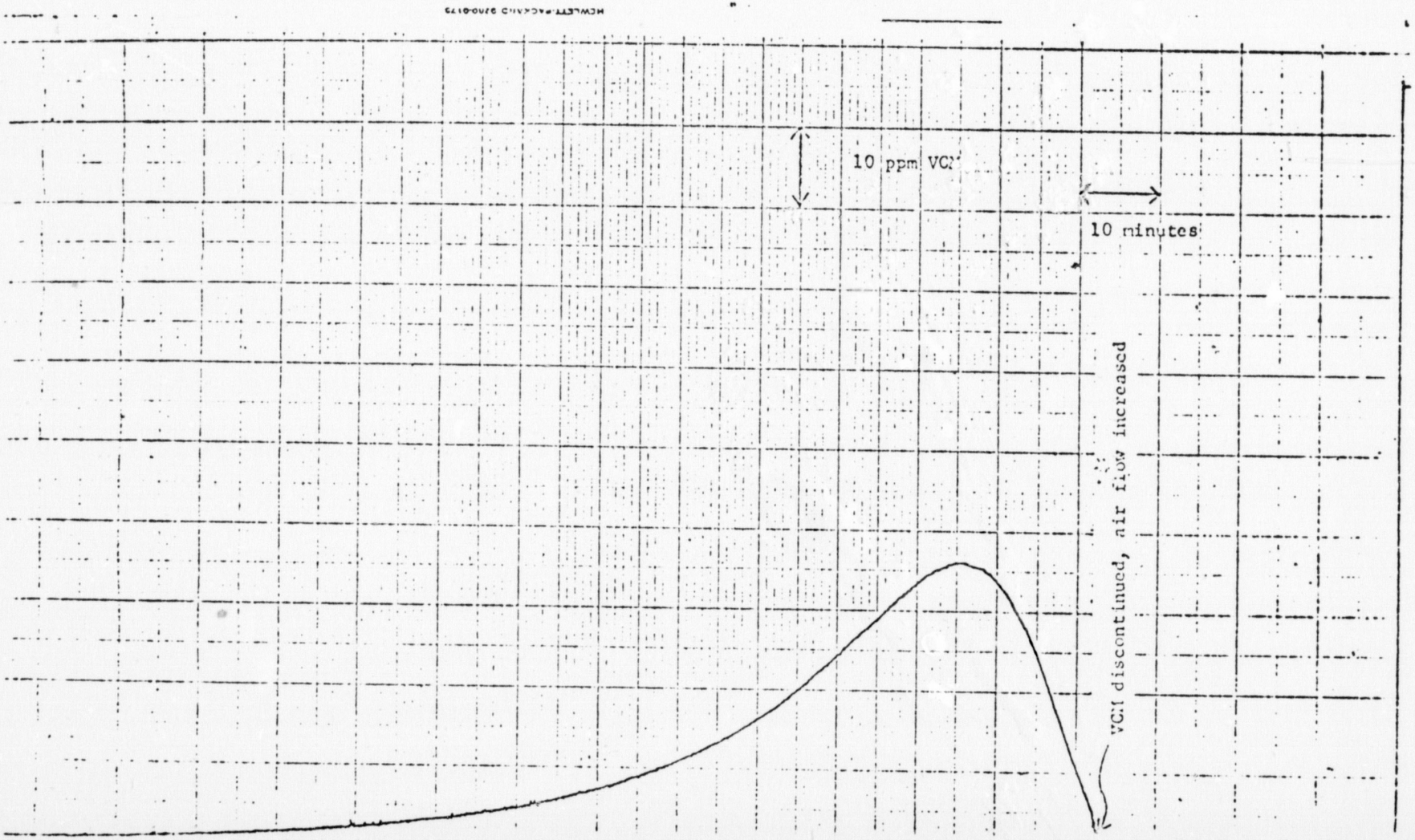
It is obvious that other chromatographic principles can be used to further decrease the desorption time of VCM on carbon. For example, higher flows in combination with heat could potentially lead to very rapid removal of the VCM from the carbon.

(iv) Performance of Cartridge after Repeated Adsorption-Desorption of Vinyl Chloride

Based on chromatographic principles, the cartridges used to adsorb vinyl chloride should be reusable once desorption has been carried out. To verify this, a 3-inch cartridge was exposed to vinyl chloride and desorbed at 64 liters/minute. This was repeated twice. This "regenerated" cartridge was then exposed to VCM at 100 ppm and 32 liters/minute. It was found that the efficiency to absorb vinyl chloride monomer was the same as found for a new cartridge. The breakthrough times for the regenerated cartridge were T_1 = 35 minutes and $T_{\frac{1}{2}}$ = 45 minutes. These values are identical to those values reported in Table 2 for a new 3-inch cartridge.

Figure 3

Desorption of VCM from 3-inch Welsh Cartridge at 64 liters/minute Clean Air



Appendix I

Experimental

Vinyl Chloride Standards

Vinyl chloride monomer in air standards (19, 900, 9990 ppm) were obtained commercially from Precision Gas Products, Inc., Rahway, New Jersey 07065.

Cartridges-Canisters

All cartridges and canisters were obtained commercially from the suppliers already mentioned. All devices were tested as received.

Make-Up Air

In order to attain the high flow rates necessary, in-plant compressed air was used to dilute the vinyl chloride standards. This air stream was purified by means of an oil filter and a 9-inch (length) by 5-inch (diameter) carbon bed. The relative humidity of make up air after passage through the carbon was 25% at 74°F being in air probably had relative humidity of ca. 60%.

Flowmeters

All streams were metered with F. & P. Co. precision bore flowmeters.

Vinyl Chloride Analyzers/Recorder

The Century Organic Vapor Analyzer used in the initial phases of this work was purchased from Century Systems Corp., Arkansas City, Kansas 67005. The Bendix Total Hydrocarbon Analyzer (Model 8101) was purchased from Bendix, Ronceverte, W. Va. 24970. A Hewlett-Packard Model 7101B Strip Chart recorder was used to display the outputs from the Century and Bendix instruments.

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GENERAL DYNAMICS

Pierre Laclede Center, St. Louis, Missouri 63105

17 July 1974

Mr. Julius Jimeno
Occupational Safety and Health Administration
U.S. Department of Labor
1726 M Street, N.W.
Room 200
Washington, D.C. 20210

Dear Mr. Jimeno:

At the conclusion of our testimony at the vinyl chloride standard hearings on 9 July we were requested to provide backup information regarding our impact statement.

In accordance with that request we are enclosing a summary of messages received from our various divisions and subsidiaries which anticipate a potential impact.

Yours very truly,

D. R. Lavalette

David R. Lavalette, Corporate Manager
Safety & Environmental Health

DRL/jh

Enc.

GENERAL DYNAMICS

SUMMARY OF DIVISION AND SUBSIDIARY MESSAGES
REGARDING IMPACT OF POTENTIAL NON-AVAILABILITY
OF POLYVINYL CHLORIDE MATERIALS

FREEMAN COAL MINING COMPANY:

"We use PVC primarily in belt conveyors and in some instances as in power cables.

STROMBERG-CARLSON:

"If the OSHA Proposed standard for exposure to vinyl chloride monomer is set so restrictive as to prevent or severely limit the manufacture of such compounds, the effect would be to suspend or severely curtail manufacturing operations at Stromberg Carlson Corporation.

The reason for this is that PVC is the only acceptable insulation for the majority of our wire and cable at this time.

During the period when a shortage of petroleum threatened to limit the supply of PVC, an extensive search was made for substitute material and no direct substitute could be found.

Our inability to supply our customers (The Independent Telephone Industry) with wire and cable and products using wire and cable would also in turn cause a deterioration of the telephone communication systems throughout the Country. All other manufacturers and suppliers of such equipment would be similarly affected. No additional telephone service could be provided and repairs and maintenance of existing systems would be severely limited.

MATERIAL SERVICE:

"PVC not used in our products or processing. Shortage will affect our suppliers of manufactured products using PVC in conveyor belt, power cable and pipe.

Assume manufacturers can furnish SBR or Neoprene rubber belting and power cable and steel pipe - subject to availability."

CONVAIR DIVISION:

"Polyvinyl chloride materials are used extensively in our space launch vehicles, particularly in the electrical harnesses and systems. These materials are also used extensively in packaging and in electrical and plumbing facilities throughout the plant.

Complete shut-off would require a major effort to rewrite specifications for alternate materials and would further require extensive testing to qualify such materials for high reliability usage in our space launch vehicles. This would stop production in those areas where the materials are used and would delay delivery of launch vehicles currently planned for high priority national programs.

for NASA, the Defense Department and the Communication Satellites.

Substitutions for packaging materials would be possible without significant impact. Substitutions for electrical and plumbing facility materials would be possible but would impact our plant maintenance. Our materials and processes people are currently reviewing over 600 specifications to further identify impact."

FORT WORTH DIVISION:

"Impact on our operations of complete shut off of polyvinyl chloride materials would be extremely severe. Some of the more obvious items utilized by us which contain PVC include electric wire and cable, sleeving, tubing, films, fabrics and coatings. We know of no substitute raw materials and/or equipment which would be readily available. Therefore, we would expect a considerable cost and schedule impact if the supply of PVC materials were completely shut off."

ELECTRIC BOAT DIVISION:

"Complete shutoff of PVC materials used at Electric Boat Division expected to have serious impact on shipyard construction and maintenance programs. Largest single area of impact is shipboard electrical wiring. Overhaul and first flite 688 class construction designs require PVC jacketing on approximately sixty percent of all wiring. Designs for later contracts like Trident and second flite 688 class anticipate PVC jacketing ninety percent all wiring. No acceptable substitutes are contained in present shipbuilding specifications. PVC also used in furniture, piping systems, and many parts of purchased assemblies. Acceptable substitutes are not always readily available. General conclusion is whenever PVC is presently specified and used, it has been chosen because it is the only acceptable product or the most cost effective product available. Available substitutes will be more expensive and will probably be difficult to procure as a result of broad based commercial competition. Where acceptable substitutes are not presently available, new product qualification testing will be costly both in terms of dollars and time. Schedules are expected to be adversely affected by a total shutoff of PVC materials, or even a significant reduction in the number of producing suppliers. Early notice of decisions will permit new designs to compensate in advance for non-availability of PVC materials."

STROMBERG-DATAGRAPHIX, SAN DIEGO:

"PVC material is widely used in many items procured by Datagraphix for use in our equipment. Major usage is in heat shrink tubing, wire coating, plastic fittings and chemical trays for film processors.

Chemical trays - Other plastic and metal materials are readily available at a significantly higher cost, however, a change over would take some time and delivery of equipment could be impacted if the change was not planned in advance.

Wire and tubing - There are ample UL listed types that do not use PVC that can be substituted; the problems would be lead times, inventory control of the new items and probably higher costs.

Fittings - Brass fittings can be readily substituted at higher price."

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February 20, 1974

2. Industrial BIO-TEST Oral Progress Report

As copies of the detailed quarterly progress report had been earlier distributed to members of the Technical Task Group on Vinyl Chloride Research, the oral presentation was primarily a summary report, augmented by slides illustrating the physical

facilities set up for this project. The tabular data presented by Mr. Schadeberg on his slides is reproduced in Appendix A to these minutes.

It was suggested that, in future progress reports, the summary tables, as exemplified by this appendix, be furnished in the copies provided for general distribution to the group, and that the detailed data be filed with MCA for the record, but not included in the general reports. Mr. Schadeberg agreed to follow this format in the future.

The mortality revealed in Table I was discussed at some length. Members were somewhat relieved to note the improving picture, particularly with respect to losses from hamster cannabilism. It was agreed not to try to replace any animals lost to date, on the grounds that the stresses introduced by such disturbances to established orders of social dominance in the populations in any given cage would tend to perpetuate the earlier situation.

The reduced weight gain of the TE-IV Group rats (those with bedding and food in the cages during exposures) was noted, but the significance of this observation, if any, could not be determined. This group will be observed carefully for evidence of systemic toxicity.

3. Report of European Research

Dr. Maltoni repeated his presentation of the results of the European research programs, and the description of on-going experiments, that he had earlier presented to the OSHA hearing on February 15 in Washington. An updated version of his report, with the inclusion of the tabular data that appeared on his slides, will, upon receipt, be distributed as a separate mailing.

The only experiment that is now essentially complete is the initial series of exposures of Sprague-Dawley rats to levels of 10,000 ppm VCM down to 50 ppm. With the exception of the highest exposure group for which early mortality limited the incidence of such cancers, the rats showed a dose-related incidence of angiosarcomas of the liver, as shown in the following table:

...k Group on
... Meeting

Rats with liver angiosarcomas	Number of rats exposed
6	69
11	72
9	74
7	67
2	67
0	64

...osure of Sp... ...ue-Dawley rats to vinyl
...ly, five d... ...s a week, for 12 months.
...omas were ... ...served in controls or
...ly to 2500 p.p... ... vinyl acetate.

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...om Tabers... ...-Cooper Association on the

...istribute... ...opies of an updated progress
... these mi... ...es as Appendix B). They
... present ... ...mat and state, their
...cts a dis... ...ation of death and is not

intended to indicate the existence or non-existence of any specific risk or excess mortality in any category.

8. Tabershaw-Cooper Associates Proposals for Expanded Epidemiological Research

Dr. Tabershaw summarized his pending proposals for new studies involving (1) intensifying their efforts to trace missing cases, (2) clinical study of suspect deaths in the mortality study, and (3) undertaking a prospective epidemiology study among high-risk segments of the worker population. He indicated that he would soon be submitting formal proposals for these projects. (Prior to the publication of the present minutes, these proposals were received and distributed to the Technical Task Group by MCA under date of March 12, 1974.)

9. Establishment of a Vinyl Chloride Pathology Register

The full task group endorsed the recommendations of the Research Coordinators that the University of Michigan be requested to reactivate and expand their acro-osteolysis registry program to include all categories of occupational illnesses specifically related to vinyl chloride exposure. They requested that MCA pursue this matter with the University, and if necessary, to seek other agencies willing to perform such a service.

10. Report of the Working Group on Analysis and Monitoring

The report of the group, in the absence of its chairman, was read, and is attached to these minutes as Appendix C. The tenor or the resulting discussion was that member companies should submit their candidate analytical and monitoring methods for consideration by the working group as soon as possible. The latter should attempt to draft its recommendations in format that provided the maximum flexibility of implementation, as long as the methods employed met appropriate standards of sensitivity, selectivity, and precision.

11. Report of the Working Group on Medical Surveillance

The recommendations of this group have been incorporated in the work practices draft submitted by that group (see the following item in these minutes). It was pointed out that the recommended surveillance is required to document the effectiveness

of the environmental controls programs. It was the opinion of the working group that liver function tests are of borderline significance and value as a predictor of the development of angiosarcomata.

12. Report of the Work Practices Working Group

The interim draft of their report was presented, with requests for suggestions as to its improvement. It was recommended that this draft (distributed to the Task Group on March 11, 1974), after review and discussion and possible revision, be presented informally to NIOSH as the personal recommendations of members of the working group now on the NIOSH consultants panel.

13. Future Meeting Schedule

In view of the uncertainties surrounding the timing of future federal agency activities relating to the vinyl chloride problem, the Task Group left the time of the next meeting subject to the call of the chairman.

Respectfully submitted,

Kenneth D. Johnson

Kenneth D. Johnson, Ph.D.
Secretary
Technical Task Group on Vinyl
Chloride Research

KJD:mb
Minutes Subject to Approval
April 8, 1974

Distribution:
- Technical Task Group on Vinyl Chloride Research
- Attendees
- Dr. Tiziano Garlanda

Appendix A

Industrial BIO-TEST Laboratories, Inc.

TABLE I

TEST MATERIAL: Vinyl Chloride (Ethylene Derived)

Chronic Vapor Inhalation Toxicity Study - Albino Rats, Albino Mice, and Golden Hamsters

Summary of Mortality Data

Total Number of Mortalities at End of Month

ALBINO RATS

Month No.	Control M	Control F	T-I M	T-I F	T-II M	T-II F	T-III M	T-III F	T-IV M	T-IV F
1	0	0	0	0	0	0	0	0	-	0
2	0	0	2	0	1	0	0	0	-	0
3	0	1	2	0	1	0	1	4	-	0
4	0	2	4	1	3	0	1	4	-	0
5	2	3	5	1	6	1	3	4	-	0

ALBINO MICE

Month No.	Control M	Control F	T-I M	T-I F	T-II M	T-II F	T-III M	T-III F
1	0	0	0	0	0	0	0	0
2	0	0	0	3	0	0	0	0
3	2	0	6	4	3	0	1	0
4	5	1	19	5	4	1	4	7
5	8	2	26	6	4	2	8	7

GOLDEN HAMSTERS

Month No.	Control M	Control F	T-I M	T-I F	T-II M	T-II F	T-III M	T-III F
1	5	4	4	4	7	5	2	5
2	8	11	10	12	9	16	10	11
3	8	13	12	14	12	18	14	13
4	9	15	16	16	15	19	15	13
5	9	18	16	16	16	20	15	15

TABLE III

TEST MATERIAL: Vinyl Chloride (Ethylene Derived)

Chronic Vapor Inhalation Toxicity Study - Albino Rats, Albino Mice, Golden Hamsters

Summary of Mean Body Weight Values - Mice

Group and Test Level	Sex	Mean Body Weights at Week Indicated (grams) 0	1	2	3	4	8	12	Total Weight Gain (grams/animal)
Control	M	35	36	37	39	40	41	43	8
	F	29	29	29	30	32	33	35	6
TE-I	M	33	34	35	37	39	39	43	10
50 ppm	F	30	29	31	31	34	34	37	7
TE-II	M	36	37	37	40	40	38	42	6
200 ppm	F	28	29	29	31	31	34	35	7
TE-III	M	36	35	35	38	38	40	43	7
2500 ppm	F	28	28	27	30	31	34	34	6

Industrial BIO-TEST Laboratories, Inc.

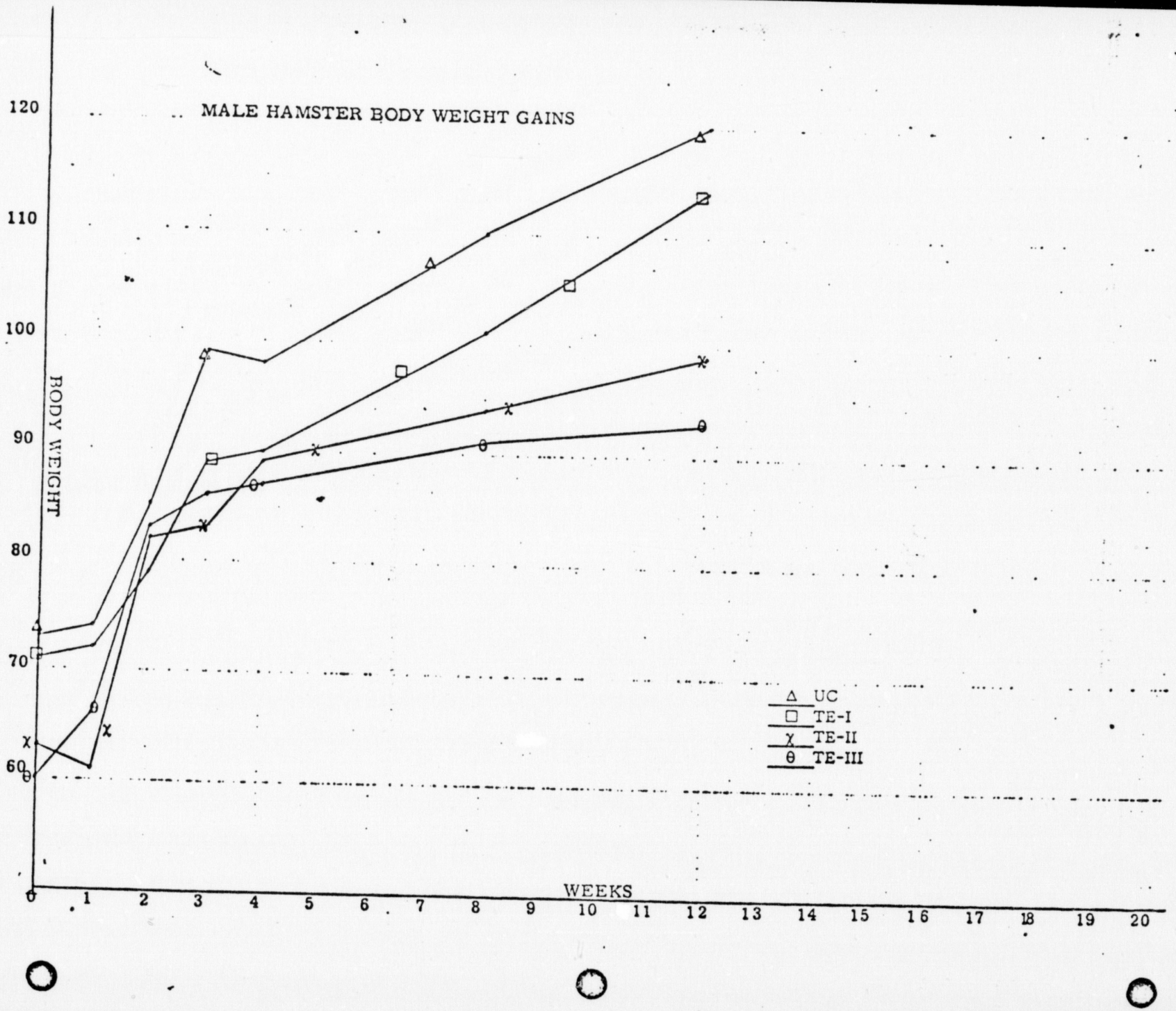
TABLE IV

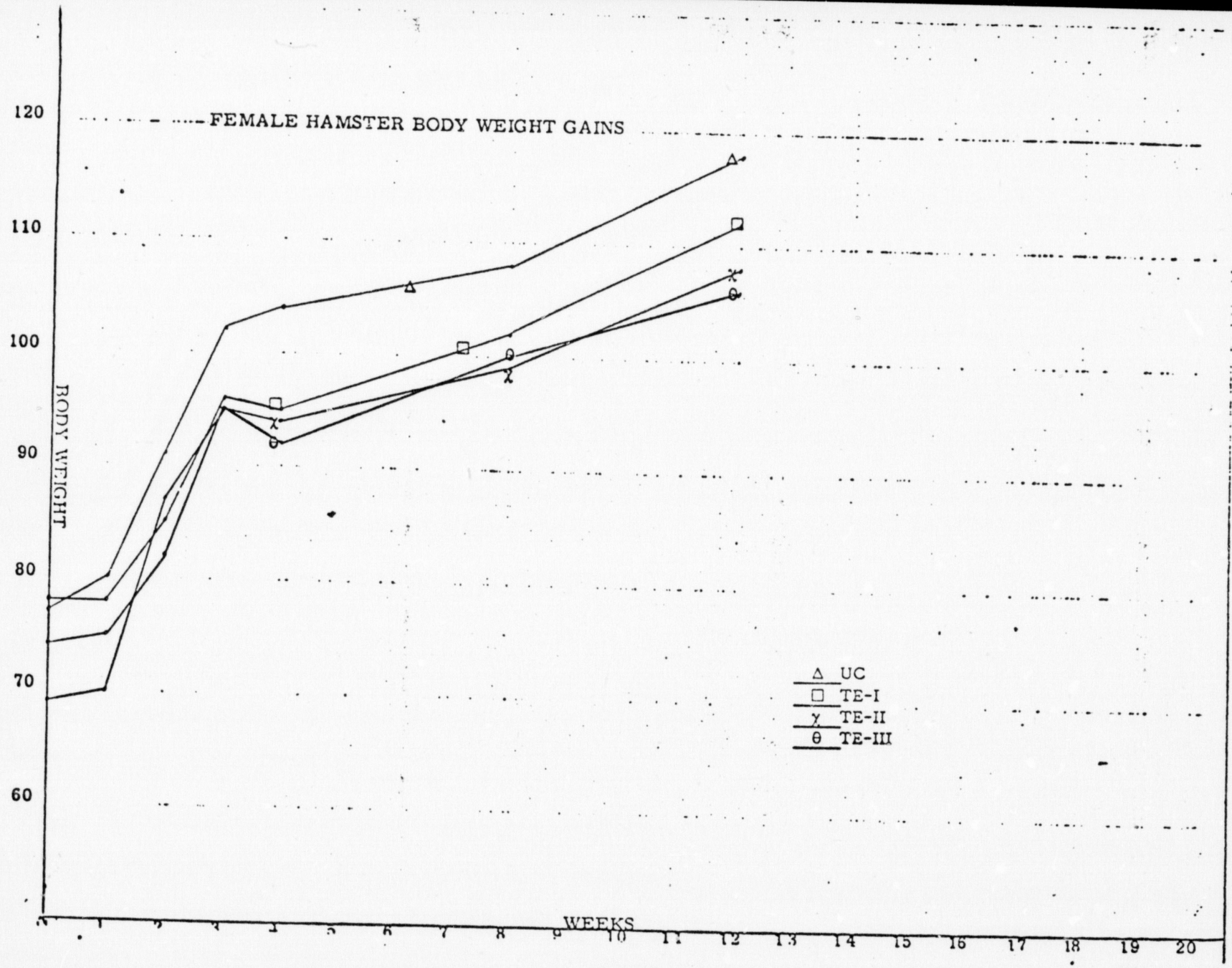
TEST MATERIAL: Vinyl Chloride (Ethylene Derived)

Chronic Vapor Inhalation Toxicity Study - Albino Rats, Albino Mice, Golden Hamsters

Summary of Mean Body Weight Values - Hamsters

Group and Test Level	Sex	Mean Body Weights at Week Indicated (grams) 0	1	2	3	4	8	12	Total Weight Gain (grams/animal)
Control	M	73	74	86	99	98	110	120	47
	F	77	80	91	102	104	108	118	41
TE-I	M	71	72	79	89	90	101	114	43
50 ppm	F	78	78	85	96	95	102	112	34
TE-II	M	63	61	82	83	89	94	99	36
200 ppm	F	74	75	82	95	94	99	108	34
TE-III	M	60	66	83	86	87	91	93	33
2500 ppm	F	69	70	87	95	92	100	106	37


MALE HAMSTER BODY WEIGHT GAINS
BODY WEIGHT
120
110
100
90
80
70
60
WEEKS
0 1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 19 20
Δ UC
□ TE-I
χ TE-II
θ TE-III


FEMALE HAMSTER BODY WEIGHT GAINS
120
110
100
90
80
70
60
BODY WEIGHT
WEEKS
0
1
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20
Δ UC
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χ TE-II
θ TE-III

Appendix B

VINYL CHLORIDE EPIDEMIOLOGY STUDY

PROGRESS REPORT: FEBRUARY 20, 1974

This is a report of a study in progress. Its purpose is to characterize the mortality experience of vinyl chloride workers up to the end of 1972. The study began in mid 1973 and consequently no attempt was made to obtain mortality data for 1973.

The study population consists of individuals who worked for at least one year in a job involving exposure to vinyl chloride before December 31, 1972, and was obtained in the following way.

We were supplied by the Manufacturing Chemists Association with the names of 43 North American plants involved in the manufacture of vinyl chloride or polyvinyl chloride. Four of these, which had begun operation after 1968, were ruled out of the study because of the brevity and recency of the exposures. Of the remaining 39, data reduction has been completed for 26, and data are in hand and being coded for six more. Of the remaining plants, four are now forwarding data to complete the required records on their workers. Study of one has been deferred because it stopped producing vinyl chloride 7 years ago, two others remain to be visited. Data are also in hand, but not yet processed, for a group of European plants.

In every plant, information on eligible workers as defined above was obtained for as far back as each plant's personnel records extended. Those who were not still employed, and who were not known from plant recordes to be either alive or dead, were traced by mail

or by a retail credit check to determine their vital status as of the end of 1972. The following tables, with the exception of the mortality data, deal with the 26 plants for which coding and key-punching are complete.

Table 1 shows the follow-up status of the study population of 4415 workers, 89 percent of whom have been traced. The 507 unknowns are still under active follow-up.

In any study in which follow-up is incomplete, it is important to examine the differences between those who have been found and those who have not. Tables 2 and 3 show the lengths of employment in exposed jobs by date of birth for the two groups. The median birth date is 1934 for those on whom follow-up is complete, and 1933 for those not yet found. The median duration of exposure is 69 months in the first group and 45 months in the second.

Similarly, Tables 4 and 5 show length of employment in exposed jobs versus the year in which exposure began for the found and not-found groups. The median date on which exposure began was 1963 for the first group and 1960 for the second.

In summary, those not yet found have about the same birth dates as those found, but have a much shorter exposure which began at an earlier age.

One interesting fact revealed by Table 2 is that there were 259 workers in the study with 20 years or more exposure and 658 with 15 years or more. These provide an excellent data base for prospective study.

Table 6 shows a distribution by selected causes for 131 deaths for whom death certificates were obtained. Certificates were included in this table even if the plants from which they came had not yet been processed. For comparison, the corresponding distribution of all U.S. male deaths in 1967 is shown. Because the age distribution of the study population is not the same as that of all U.S. males, and because the number at risk in the study population is not considered, this table is only descriptive. The main point of the table is to show that for rare conditions such as cancers of specific sites, the number of cases appearing in the study is too small to enable definite conclusions to be drawn. In order to investigate such rare conditions, the deaths must be studied in more detail than is possible from information contained in the death certificate itself.

When the remainder of the study plants have been processed, an estimated 150 - 200 more deaths will be found, since some of the oldest plants are in the group still being processed. At that time, calculations of the risk of death by broad cause groups will be made.

In summary, this study of the mortality of workers occupationally exposed to vinyl chloride is still incomplete. Of the 5000 to 5500 workers ultimately to be included, 4415 have so far been identified. So far the vital status of only 89% of these has been determined. Reports are still coming in on some of the oldest plants, so that the 120 deaths so far known will probably rise to 300 or more as data accumulate.

Because rare tumors of the kind suspected in recent weeks as being associated with VCM are not clearly identifiable in mortality statistics, a routine study of mortality ratios would not be expected to reveal them unless the incidence was quite high. For this reason this type of mortality study must be supplemented by case analyses. It should also be emphasized that the collection of identifying information on a population known to have been exposed for periods exceeding 10, 15 and 20 years provides a data base that will facilitate prospective studies.

Vinyl Chloride Epidemiology Study

Table 1

Study Population by Follow-up Status

Total	Alive	Dead	Unknown
4415	3780	128	507

Percent Follow-up - 89

February 15, 1974

Vinyl Chloride Epidemiology Study

Table 2

Distribution by Birth Date and Years of Exposure, for Members of Study Population for Whom Follow-up is Complete*

Birth Date	Total	Years of Exposure 1-<5	5-<10	10-<15	15-<20	20+
Total	3877	1774	1052	393	399	259
1890-99	10	5	2	2	1	
1900-09	90	21	18	19	17	15
1910-19	434	103	80	65	95	91
1920-29	910	236	215	135	185	139
1930-39	1103	458	366	164	101	14
1940-49	1261	883	370	8		
1950+	68	68				
Unknown	1		1			

*Duration of exposure unknown for 31 workers.

February 15, 1974

Vinyl Chloride Epidemiology Study

Table 3

Distribution by Birth Date and Years of Exposure, for Members of Study Population Not Yet Found*

Birth Date	Total	Years of Exposure 1-<5	5-<10	10-<15	15-<20	20+
Total	497	366	73		14	8
1890-99	1	1				
1900-09	22	15	2			3
1910-19	60	34	12	7	4	3
1920-29	126	91	13	10	10	2
1930-39	143	92	34	17		
1940-49	142	130	12			
1950+	3	3				

*Duration of exposure unknown for 10 workers.

February 15, 1974

Vinyl Chloride Epidemiology Study

Table 4

Distribution by Year Exposure Began
and Number of Years of Exposure,
for Members of Study Population
on Whom Follow-up is Complete*

Year Exposure Began	Total	Years of Exposure 1-<5	5-<10	10-<15	15-<20	20+
Total	3877	1774	1052	393	399	259
1940-49	290	49	34	26	43	138
1950-59	1020	163	131	249	356	121
1960-69	2126	1121	887	118		
1970-71	441	441				

*Duration of exposure unknown for 31 workers.

February 15, 1974

Vinyl Chloride Epidemiology Study

Table 5

Distribution by Year Exposure Began
and Number of Years of Exposure,
for Members of Study Population Not Yet Found*

Year Exposure Began	Total	Years of Exposure 1-<5	5-<10	10-<15	15-<20	20+
Total	497	366	73	36	14	8
1940-49	73	50	8	8		7
1950-59	172	101	30	26	14	1
1960-69	239	202	35	2		
1970-71	13	13				

*Duration of exposure unknown for 10 workers.

February 15, 1974

Table 6

Percentage Distribution by Selected Causes for 131 Deaths in Vinyl Chloride Workers, and Percentage Distribution for the Same Causes in U.S. Males, 1967

Cause of Death	U.S. Males 1967	Vinyl Chloride Workers
All Causes	100.0%	100.0% (131)
Malignant neoplasms (140-205)	16.2	19.8 (26)
Buccal cavity and pharynx (140-148)	0.5	2.3 (3)
Digestive organs and peritoneum (150-159)	4.9	3.1 (4)
Liver (156)	0.2	0.8 (1)
Respiratory system (160-164)	4.6	6.9 (9)
Urinary organs (180,181)	0.9	0.8 (1)
Brain (193)	0.4	2.3 (3)
Leukemia and aleukemia (204)	0.8	0.8 (1)
Lymphosarcoma and other neoplasms of lymphatic and hematopoietic tissues (200-203, 205)	0.9	1.5 (2)
Major cardiovascular-renal diseases (330-334, 400-468, 592-594)	53.0	47.3 (62)
Vascular lesions affecting CNS (330-334)	8.9	3.8 (5)
Arteriosclerotic heart disease (420)	33.0	35.9 (47)
Hypertensive disease (440-447)	2.6	1.5 (2)
Cirrhosis of liver	1.7	1.5 (2)
Motor vehicle accidents (E810-E835)	3.6	6.9 (9)
Other accidents (E800-E802, E840-E962)	3.8	6.9 (9)
Suicide (E963, E970-E979)	1.5	3.8 (5)
Other	20.2	13.7 (18)

Appendix C

Subcommittee on Analytical and Monitoring Methods
Task Group on Vinyl Chloride
Manufacturing Chemists Association
Volunteered at meeting of January 25, 1974

B. Barnes, ICI
Z. G. Bell, PPG
R. Henderson, Olin
H. R. Hoyle, Dow (volunteered by T. R. Torkelson)
F. Kennedy, Continental Oil

Subsequent to the meeting, T. Aalto, Tenneco, has volunteered to serve on the subcommittee.

B. Barnes, Z. G. Bell, R. Henderson and F. Kennedy met on January 25, 1974. At that meeting and in subsequent conversations it was agreed that the role of the subcommittee should be to:

I. Review literature on methods of analysis for VCM.

II. Solicit information from contributors to the M.C.A. vinyl chloride projects on their experience with various procedures for sampling work environments and analyzing the samples.

III. Evaluate procedures for sampling and analyses.

IV. Recommend procedures for sampling and analyses.

It was agreed that there are analytical methods of sufficient sensitivity to detect VCM in air in the concentration range of one to 50 parts per million using reasonable air samples. Flame ionization, infrared, and pyrolysis followed by measurement of conductivity of water in which chloride is trapped are detection methods that can be used.

Members of the M.C.A. vinyl chloride project will be requested to submit to Dr. Henderson information on procedures for monitoring and analysis for VCM.

The members of the subcommittee will meet after information is received from member companies and circulated for evaluation.

Richard Henderson

Richard Henderson
Chairman, Subcommittee on
Analysis and Monitoring

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MANUFACTURING CHEMISTS ASSOCIATION

1825 CONNECTICUT AVENUE, N.W. WASHINGTON, D.C. 20009 (202) 483-6126

April 17, 1974

To: TECHNICAL TASK GROUP ON VINYL CHLORIDE RESEARCH

Subject: Report on Conference with Industrial BIO-TEST Laboratories, April 15, 1974

Gentlemen:

As a result of the telephoned information received by MCA from Industrial BIO-TEST personnel last week, a conference at the Northbrook, Illinois offices of IBT was set up at which an oral status report on the chronic inhalation studies with vinyl chloride was presented to MCA, industry and federal agency representatives.

Those in attendance, and their affiliations, are shown below:

Z. G. Bell, Jr.	PPG Industries, Inc.
A. S. Cummin	Borden, Inc.
E. J. Fairchild	NIOSH
J. W. Goode	Industrial BIO-TEST Laboratories
D. E. Gordon	Industrial BIO-TEST Laboratories
W. D. Harris	UNIROYAL, Inc.
M. N. Johnson, M.D.	The B. F. Goodrich Company
Flynt Kennedy	Continental Oil Company
M. L. Keplinger	Industrial BIO-TEST Laboratories
R. J. Kociba	The Dow Chemical Company
H. L. Kusnetz	Shell Oil Company
D. V. Lassiter	OSHA
W. E. Rinehart	Ethyl Corporation
W. M. Smith	Air Products & Chemicals, Inc.
J. F. Stara	U. S. Environmental Protection Agency
T. R. Torkelson	The Dow Chemical Company
R. N. Wheeler	Union Carbide Corporation
B. M. G. Zwicker	B. F. Goodrich Chemical Company
R. F. Blewitt	MCA
A. C. Clark	MCA
K. D. Johnson	MCA

Dr. Goode first presented the mortality data and gross observations, and then Dr. Gordon described the pathology observed in the mice. The data, as presented, are shown in the attached tables. There are apparent discrepancies between the total mortality data and the detailed listing of recent deaths. These will be resolved. No test-related pathology has yet been noted in any animals other than mice.

Dr. Fairchild, of NIOSH, reported that Dr. David Rall, of HEW, had appointed a committee to coordinate departmental toxicology and related programs, and that under this committee there has been named a subcommittee to develop a "white paper" on guidelines for research to determine "socially acceptable" levels of risk. A report from this subcommittee, which will cover both philosophical and statistical considerations, is expected in the next few weeks.

Dr. Stara expressed the view that the vinyl chloride problem presented industry with a unique opportunity to seek to demonstrate a "no effect level" of a human carcinogen with a sensitive animal model of well-documented validity.

Dr. Lassiter expressed a guarded judgment that the mouse data reported by IBT would not lead OSHA to modify the emergency temporary standard, but that it might well cause OSHA to accelerate the rule-making process for the development of a permanent standard.

Dr. Lassiter also reported briefly on the retrospective morbidity and mortality studies being conducted by NIOSH/CDC at nine or ten locations (six companies) around the country. No epidemiological data were presented.

In an afternoon session of the Research Coordinators, it was agreed that the exposure of the mice in the present project should be terminated at nine months, but that exposures of both rats and hamsters should proceed for the initially-planned twelve months.

It was further moved, seconded and carried that the chairman appoint a group to begin investigating the cost and availability of facilities of new experiments to be conducted at lower levels of exposure, with larger groups of animals. He was asked specifically to seek to encourage the National Center for Toxicological Research, at Pine

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Bluff, Arkansas, to offer to undertake such a program. Details of the test protocol are to be left open until after the Rall committee makes its report available.

The Research Coordinators asked MCA to call a meeting of the Technical Task Group on Vinyl Chloride Research as soon as the report of Tabershaw-Cooper Associates on their epidemiological study reaches them.

A copy of the MCA news release reporting the Industrial BIO-TEST mouse data has been sent to you in this morning's mail.

Sincerely,

Kenneth D. Johnson, Ph.D.
Secretary
Technical Task Group on
Vinyl Chloride Research

KDJ:mb

Tables Attached

cc : Mr. A. W. Barnes
D. P. Duffield, M.D.
Dr. Tiziano Garlanda
Vinyl Chloride Management Contacts

TABLE

Mouse Mortality Data

DATE	GROUP							
	Controls		TI		TII		TIII	
	F	M	F	M	F	M	F	M
February 10, 1974	2	8	6	26	2	4	7	8
April 12, 1974	5	11	9	31	13	7	13	18

Industrial BIO-TEST Laboratories, Inc.
1810 FRONTAGE ROAD
NORTHBROOK ILLINOIS 60062

TABLE II

Summary of Tumors in Vinyl Chloride Exposed Mice
IBT Number 663-03222-MCA

Group, Animal No., Sex	Histopathologic Findings			Date of Death
	Lung	Liver	Skin	
T-I				
1023 Male	AA (s)*	HS (s)**		
1110 Female	AA (s), Metastatic Skin Tumor		EC*** (para-aural)	3-20-74
1119 Female		HS (s)		3-24-74
1200 Female	Metastatic Skin Tumor		EC & MC+ (Femoral region)	3-30-74
T-II				
1347 Female	Metastatic Skin Tumor		EC(Abdomen)	3-5-74
1309 Female		HS (s)		3-18-74
1889 Male	AA (s)	HS		3-25-74
1382 Female		HS (s)		4-2-74
1350 Female	AA (s)	HS (s)		4-5-74
1299 Female	AA (s)		EC & MC (Inguinal Region)	4-9-74
T-III				
1538 Female	AA (s)	HS (s)		3-6-74
1532 Female	AA (s)		LSC	3-7-74
1562 Female	AA (s)	HS (s)		3-[illegible]-74
1598 Female	AA (s)	HS (s)		
1405 Male	AA (s)	HS (s)		2-[illegible]-74
1494 Male	AA (s)	HS (s)		3-22-74
1574 Female	AA (s)	HS (s)		3-22-74
1531 Female		HS (s)	EC & MC (Femoral region)	3-23-74
1588 Female		HS (s)		3-25-74
1593 Female	AA (s)	HS (s)		3-30-74
1505 Female	AA (s)	HS (s)		4-3-74
1565 Female	AA (s)	HS (s)		4-3-74
1409 Male	AA (s)	HS (s)		4-5-74
1543 Female	AA (s)	HS (s)		4-5-74
1544 Female	AA (S)	HS		4-5-74
1546 Female	AA (s)	HS (s)	MC (Axillary region)	4-5-74
1535 Female	AA (s)	HS (s)		4-7-74
1432 Male	AA (s)			4-8-74
1477 Female	AA (s) Metastatic Skin Tumor?	HS (s)	EC (Para-Aural) MC (Abdomen) EC(Pelvic region)	4-9-74

TABLE III

Industrial BIO-TEST Laboratories, Inc.
1810 FRONTAGE ROAD
NORTHBROOK, ILLINOIS 60062

Summary of Mortality & Tumor Data Among Vinyl Chloride Mice
IBT Number 663-03222 (MCA)

Group	No. Mortalities with Neoplasms	Location of Tumors			
		Lung	Liver	Skin	Metastasis → Lv
T-I	4 (1 Male, 3 Females)	2(50%)	2(50%)	2(50%)	2
T-II	6 (1 Male, 5 Females)	3(50%)	4(66.7%)	2(33%)	1
T-III	19 (4 Males, 15 Females)	17(89.5%)	17(89.5 %)	3(15.8%)	

Key to Symbols

* Alveologenic Adenoma of Lung (AA)
** Hemangiosarcoma of Liver (HS)
*** Epidermoid Carcinoma of Skin (EC)
\+ Mammary Carcinoma of Skin (MC)
⊕ Lymphosarcoma of Thymus
(s) Multiple tumors of indicated type

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INTERIM RESULTS OF EXPOSURE OF RATS, HAMSTERS AND MICE TO VINYL CHLORIDE

M. L. Keplinger, Ph.D.,
J. W. Goode, Ph.D.,
D. E. Gordon, D.V.M., Ph.D.
and
J. C. Calandra, M.D., Ph.D.
Industrial Bio-Test Laboratories, Inc.
Northbrook, Illinois

The following data are partial results of a study in progress at the Industrial Bio-Test Laboratories, Inc. The studies are being coordinated for sponsoring companies by the Manufacturing Chemists Association. The data are very incomplete but because of their significance were first presented to EPA, NIOSH and OSHA on April 15, 1974.

In this study which was designed to complement Maltoni's studies, 100 rats, hamsters and mice of each sex (600 total per exposure group) are being exposed to either 0, 2500, 200 or 50 ppm vinyl chloride vapor 7 hours per day 5 days per week. Mice will be exposed for 9 months and kept for an additional 9 months. Rats and hamsters will be exposed for 12 months and kept for an additional 12 months.

The rats are COBS Charles River; mice are CDI Swiss Charles River; and the hamsters are Golden Syrian from the same source. Food is removed during exposure, water is present and no bedding is used at any time.

Moribund and dead animals (when possible) receive gross autopsies and are photographed. The major organs of all animals are fixed for histological examination.

Only the tissues from a few animals have been examined so far. The table shows the results to date (about 8 months exposure).

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Note that no direct numerical comparisons can be made with the control groups as they have not been selected in the same way. Only mice with tumors have been examined in the exposure groups. No tumors have been observed grossly as yet in any of the control mice.

In addition to the tumors in mice a few tumors have been observed in rats and hamsters but because of autolysis the diagnosis at this time must be considered tentative. At this time it appears that angiosarcomas may have been observed in one male hamster and one male rat exposed to 2500 ppm and in one female rat exposed to 200 ppm. No tumors have been observed in rats or hamsters exposed to 50 ppm but one male control was found to have a dermal fibroma.

These tentative data need to be confirmed and will be reported in detail when the study is completed.

INTERIM SUMMARY OF TUMORS IN MICE EXPOSED TO VINYL CHLORIDE FOR EIGHT MONTHS

EXPOSURE GROUP	NUMBER OF MORTALITIES WITH NEOPLASMS			LOCATION AND TYPE OF TUMOR			
				ALVEOLOGENIC ADENOMAS, LUNG	ANGIOSARCOMA, LIVER	ADENO SQUAMOUS CARCINOMA, MAMMARY	MAMMARY, WITH METASTASIS TO LUNGS
	TOTAL	MALE	FEMALE				
CONTROL*	0	(7)	(2)	0	0	0	0
50 PPM**	4	1	3	2	2	2	2
200 PPM**	15	3	12	12	11	3	1
2500 PPM**	30	6	24	28	28	6	1

* 7 MALE AND 2 FEMALES CONTROL MICE HAVE BEEN EXAMINED HISTOLOGICALLY. NONE HAD TUMORS IN LIVER, KIDNEYS, HEARTS OR LUNGS.

** ONLY MICE WITH TUMORS HAVE BEEN EXAMINED HISTOGOGICALLY.

MICE WERE EXPOSED 7 HOURS/DAY, 5 DAYS/WEEK.

25003

Preliminary Findings
from MCA Administered Study at
Industrial Bio-Test Laboratory

April, 1974

2503

COVINGTON & BURLING
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HARRIS WEINSTEIN
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PETER J. NICKLES
MICHAEL BOUDIN
BINGHAM B. LEVERICH
ALLAN J. TOPOL

August 15, 1974

Mr. Jules Jimeno
Occupational Safety and
Health Administration
Docket OSH-36
Room 230
1726 M Street, N.W.
Washington, D. C. 20210

Dear Mr. Jimeno:

As a supplement to the Tenneco Chemicals, Inc., testimony on June 27, 1974, and in an effort to assist OSHA further in considering an appropriate permanent standard relating to vinyl chloride, Tenneco is submitting the following additional information:

1. A statement prepared by Dr. Paul Lobo, who was a Tenneco witness at the OSHA hearing, which relates to the testimony of the Calgon representatives on July 11, 1974. This statement is Exhibit A hereto.

2. A statement prepared by Dr. Paul Lobo which quantifies theoretically the relationship between fugitive emissions and VCM exposure in a PVC plant. This statement is Exhibit B hereto.

3. A statement prepared by Mr. P. A. Ketterer, a Tenneco employee, presenting data on the accuracy and reproducibility of various personnel monitoring methods for determining VCM content in the air. It will be recalled that at the hearing Mr. Fath, a Tenneco witness, testified that personnel monitoring

could be used as a check on constant sequential area monitoring. It is essential that a personnel monitoring system, used for even this limited purpose, be as accurate as possible. This exhibit shows that the bag method is more accurate than the charcoal tube method. This statement is Exhibit C hereto.

4. Recent articles from Chemical Engineering Progress (July 1974) and Chemical Week (August 7, 1974) which describe current lead-time problems in purchasing new equipment. The necessity for an adequate lead-time to permit the purchase and installation of new equipment needed for compliance with the proposed permanent standard was the subject of testimony at the OSHA hearing. These articles are Exhibits D-1 and D-2 hereto.

5. Data on exposure levels in Tenneco's Nixon, New Jersey, plant, which is a rigid calendering plant; in Tenneco's Newton, Massachusetts, plant, which is a flexible calendering plant; and in Tenneco's Carlstadt, New Jersey, plant, which is a fabric coating from plastisol plant. These data are Exhibits E-1, E-2 and E-3 hereto.

In addition to the attached information, Tenneco will submit by August 23 all of the information which the Tenneco witnesses agreed during cross-examination to furnish.

Sincerely yours,

Allan J. Topol

Allan J. Topol
Counsel for
Tenneco Chemicals, Inc.

emf
Enclosures

EXHIBIT B

FUGITIVE VINYL CHLORIDE EMISSIONS FROM A POLYVINYL CHLORIDE PLANT -- EFFECT ON WORKER EXPOSURE LEVELS

Currently vinyl chloride monomer (VCM) emissions from Tenneco Chemicals' Burlington, New Jersey, and Flemington, New Jersey, plants amount to 3-4 percent of the VCM charged to the process. Engineering improvements, based on existing technology, can be made to reduce VCM losses to the surroundings. These comprise: (1) minimizing losses due to VCM tank car unloading; (2) the elimination of a major portion of the VCM emissions occurring during opening of the reactor via installation of improved reactor cleaning systems; (3) removal of VCM at the slurry stage, thus eliminating losses of VCM downstream in the drying, bagging and storage areas; and (4) removal of VCM from inert purge streams via the installation of activated carbon adsorption units.

Although it is impossible to determine at this point in time exactly how much VCM emissions will be reduced by implementation of these engineering improvements, it is expected that emissions will be reduced to one percent of VCM charged, the sources of most of the remaining losses being the so-called "fugitive emissions."

Fugitive emissions are those losses which occur at valves, mechanical seals, pumps, compressors, flanges, etc. To indicate the magnitude of the number of such points of loss in the Burlington, New Jersey, plant, it is estimated that the PVC plant contains approximately 1,500 valves, 15,000 flanges and roughly 50 rotary seal units.

Assuming that replacement of these components could be made with similar components less prone to leak, some of these not currently available, fugitive emissions could be reduced by perhaps a factor of two or three to 0.4 percent of the monomer charged. At Tenneco's Burlington, New Jersey, plant, where approximately 500,000 pounds of monomer are used per day, this means a loss of 2,000 pounds per day or approximately 80 pounds per hour throughout those areas of the plant where VCM is handled.

If a theoretical model is assumed consisting of a five acre area enclosed to a height of 30 feet in which one pound of VCM is uniformly distributed, the result would be the presence of 9 ppm of VCM in that theoretical atmosphere. Although in a plant site of similar dimensions diffusion through natural and mechanical movement of air would, of course, occur, the problem of dealing with even small quantities of VCM lost after all the above corrective measures are taken is apparent.

2506-A ASI Copy

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ALLAN J. TOPOL

JOHN SHERMAN COOPER
EDWIN S. COHEN
OF COUNSEL

August 23, 1974

Mr. Jules Jimeno
Occupational Safety and
Health Administration
Docket OSH-36
Room 230
1726 M Street, N.W.
Washington, D. C. 20210

Dear Mr. Jimeno:

In the testimony of the Tenneco witnesses, Messrs. Fath, Lobo and Scarito, at the OSHA hearing on June 27, 1974, several requests were made to the witnesses to submit additional information for the record. The Tenneco witnesses agreed that the information requested would be submitted.

Accordingly, attached are the following items of information which constitute Tenneco's response to the requests made at the hearing.

1. Summaries of personnel monitoring data from the Burlington resin plant and the Flemington resin plant, indicating the vinyl chloride exposures of employees by job classifications. These data are responsive to the requests of Mr. Kline on pages 710-711 and 728-729 of the transcript. Tenneco is not able to state whether the exposures on the attached tables are intermittent or general exposures because the data are based upon personnel monitoring, and data of this type do not permit a judgment to be made as to whether exposures are intermittent or general. (These data are Attachment 1 to this letter.)

2. The experimental data relating to the residual monomer content which forms the basis for the 100 ppm figure in the testimony of the Tenneco witnesses. This

experimental data was requested by Mr. Kline on page 724 of the transcript. (These experimental data and accompanying explanation are Attachment 2 to this letter.)

3. Data relating to the rate of employee turnover during the last two or three years at the various production plants of Tenneco at which there is VCM exposure. These data were requested by Mr. Kuchenbecker on page 736 of the transcript. (These data are Attachment 3 to this letter.)

4. Results of medical examinations of employees at Tenneco's VCM, PVC and processing plants which Tenneco has thus far received from National Health Services. Tenneco contracted with National Health Services to conduct these examinations, and a description of National Health Services is included with the results. This information is submitted in response to Mr. Samuels' request on page 742 of the transcript. (The information is Attachment 4 to this letter.)

5. Results of OSHA's monitoring at Tenneco facilities which have been transmitted to Tenneco. These were requested by Mr. Cottine on page 750 of the transcript. (These results are Attachment 5 to this letter.)

6. Results of EPA's monitoring at Tenneco facilities which have been transmitted to Tenneco. These were requested by Mr. Cottine on page 750 of the transcript. These results are already in the record as Exhibit 7(q).

7. A copy of Tenneco's current medical protocol. This request was made by Mr. Cottine on page 752 of the transcript. (The protocol is Attachment 6 to this letter.)

Very truly yours,

Allan J. Topol

Allan J. Topol
Counsel for
Tenneco Chemicals, Inc.

emf
Enclosures

2506-C

ATTACHMENT 2

EXHIBIT

REDUCTION OF VINYL CHLORIDE MONOMER IN POLYMER

To determine the feasibility of removing the residual vinyl chloride monomer (VCM) from polyvinyl chloride resin after the polymerization step, slurry samples from the plant were subjected to treatment in glass flasks in the laboratory, with and without stripping. Samples of the slurry were withdrawn from the flasks at specified times and analyzed for VCM content and these data are given in the attached Table I.

As can be seen from these data, reduction of the VCM content of the slurry, and hence the final resin, is feasible in the laboratory. Currently, translating of the laboratory results to plant operation is in progress.

[redacted] ATTACHMENT 2 [redacted]

EXHIBIT

TABLE I

HOMOPOLYMER RESIN

REDUCTION OF VCM IN SLURRY

LABORATORY DATA

DATE OF TEST	LAB NOTEBOOK REFERENCE	RESIN FORM	INITIAL VCM CONTENT (PPM) *	STRIPPING	VCM CONCENTRATION (PPM)* ½ Hr.**	1 Hr.**	2 Hr.**	3 Hr.**
4/18/74	DO42-102	Slurry	4663	Yes		1131	726	385
4/19/74	DO42-104	Slurry	4663	Yes		20	0	0
4/15/74	EO51-46	Slurry	485	No		35	30	25
4/15/74	EO51-46	Slurry	485	No		20	10	0
4/15/74	EO51-46	Slurry	485	No		15	5	0
4/19/75	DO42-108	Slurry	4571	Yes		1807	1300	1059
4/20/74	DO42-110	Slurry	4571	Yes		116	21	13
4/19/74	EO51-49	Slurry	2855	No		308	225	170
4/19/74	EO51-49	Slurry	2855	No		175	90	75
4/29/74	EO25-116	Slurry	385	No	0			
4/15/74	EO51-45	Slurry	820	No		275	200	190
4/15/74	EO51-45	Slurry	820	No		140	70	45
4/15/74	EO51-45	Slurry	820	No		40	20	13
4/30/74	EO25-117	Slurry	29	No	0			

* VCM concentration in slurry.

** Slurry sample from plant was treated in glass flask. Samples withdrawn at specified times and analyzed for VCM content of the slurry.

2506-D

2507

109

July 26, 1974

Ms. Joanne Goodell
Docket OSH - 36
Room 230, 1726 M Street NW
Washington, D.C. 20210

Proposed standard for Vinyl Chloride 39-FR 16896 - Hearing - Exhibit No. 43

Dear Ms. Goodell:

We want to thank you for the courtesy extended our Dr. Brecker at the time of his presentation to the Hearing on July 10th on behalf of Witco Chemical Corporation.

Since Dr. Brecker's statement as delivered at the Hearing contained a few changes from the prepared text, we are enclosing for the record two copies of his statement as delivered. We are also enclosing for the record two copies of our laboratory report on determination of Vinyl Chloride monomer in air, because this was mentioned in answer to questions after Dr. Brecker's presentation and we were requested to submit it for the record.

Accordingly, we request that the enclosed statement be included in the record and given favorable consideration in arriving at the permanent standard.

Thank you.

Sincerely,

Otto S. Kauder

Dr. Otto S. Kauder
Vice President - Research

gd/OSK

Argus Chemical Corporation
subsidiary of Witco Chemical Corporation
633 Court Street, Brooklyn, New York 11231

Telephone 212—858-5678
Cable: Catchem, N.Y.

42

2508

ARGUS CHEMICAL CORPORATION
SUBSIDIARY OF WITCO CHEMICAL COMPANY, INC.

DATE May 1, 1974 FROM Robert Bruce and Glenn Carr

TO: Mark W. Pollock

SUBJECT Determination of Vinyl Chloride Monomer Content in Air (Project # 361)

CONCLUSION:

Vinyl chloride monomer content of the mill room atomosphere is substantially less than 50 ppm. Air samples taken during mill processing of PVC compounds and at a distance within a foot and a half of the mill rollers and within 30 seconds of placing the sample on the mill showed a maximum monomer content of 8.5 ppm for one sample out of seven; the other six samples including two samples taken at the north and west sides of the mill room showed less than 1 ppm vinyl monomer.

METHOD OF ANALYSIS :

Vinyl chloride monomer is estimated by gas chromotagraphy using a Poropak S column and the flame detector. Air samples are taken using a 10 ml. gas syringe supplied by Glenco Scientific, Inc., catalog # 19925-50.

GLC CONDITIONS:

Instrument	:	Perk-Elmer 900
Column	:	1/8 inch x 3 feet stainless steel Poropak S
Injection Port	:	150° C
Column	:	90° isothermal
Flame Detector	:	180°C
Sample Size	:	10 microliters at range 10, alternation 16
Carrier Gas	:	Helium at 45 ml per min.

VINYL CHLORIDE MONOMER CALIBRATING STANDARDS

Air samples of vinyl monomer are prepared by the addition of vinyl chloride gas to rubber capped 1000 ml glass volumetric flasks. This is accomplished by passing a stream of V.C. gas from a Matheson lecture bottle through ¼ inch Tygon tubing until the tubing is purged of air. A 50 microliter Hamilton syringe is inserted into the open end of the tubing and the plunger is moved up and down to purge the needle of air. Then 10,20,30,40 and 50 microliters of V.C. gas are injected into volumetrics to give solutions of 0 to 50 ppm. The instrument is calibrated by injecting 10 ml samples of each standard and by plotting peak height against concentration in ppm. The following are results of the calibration procedure:

ARGUS CHEMICAL CORPORATION
SUBSIDIARY OF WITCO CHEMICAL COMPANY, INC.

DATE May 1, 1974 FROM Robert Bruce and Glenn Carr

TO: Mark W. Pollock

SUBJECT Determination of Vinyl Chloride Monomer Content in Air (Project # 361) Page - 2 -

Vinyl Chloride Gas Conc. Microliters /liter	Peak Height
0	
10	0.88
20	1.75
30	2.72
45	3.40
50	4.20

A plot of peak height vs. concentration results in a straight line (see attached calibration graph).

ANALYSIS OF MILL ROOM AIR SAMPLES

Samples of Geon 103EP, Diamond 40, Diamond 450, Eleanora 451, Rucon B34, and Geon 120 using formulation

Resin	100
DOP	45
Drapex 6.8 epoxide	5
Stabilizer Mark LL	2
Stearic Acid	0.25

were run on mill at 150°C and air sample taken within 30 seconds. The following results were obtained

Sample	Peak Height	ppm V. C. M.
Diamond 40	0.78	8.5
Geon 103EP	0.10	approx. 1.0
Diamond 450	Trace	Trace
Eleanora 451	Trace	Trace
Rucon B34	Trace	Trace
Geon 120	Trace	Trace
North Side of Mill Room		1.0
West Side of Mill Room		1.0

c.c. Mr. W. E. Leistner; Mr. W. E. Setzler; Mr. Seymour Cohen; Mr. Nirmal S. Jain; Dr. Otto S. Kauder; Dr. Lawrence R. Brecker Mr. S. Gladstone; Dr. L. B. Nelson; Dr. Motonobu Minagawa; Mr. Leon Dubru Dr. Walter Weber; Mr. Alfred G. Williamson; Mr. S. C. Anand; Dr. Werner Mitteldorf

gd/GC,RB

NO. 341-5¼ DIETZGEN GRAPH PAPER
5 X 5 PER HALF INCH

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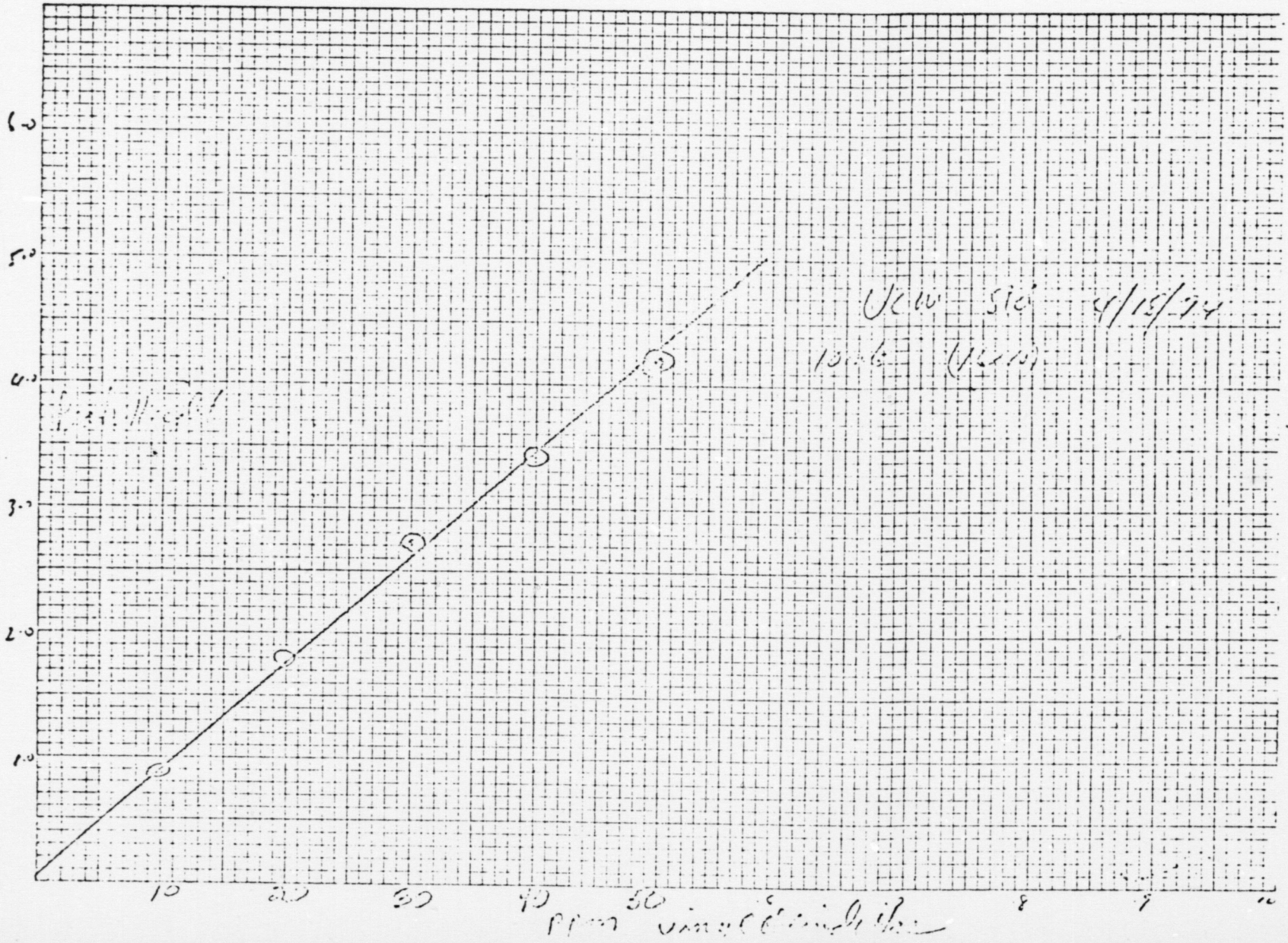
MADE IN U. S. A.



SUBSIDIARY OF WITCO CHEMICAL COMPANY, INC.

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c.c.. Mr. W. E. Leistner; Mr. W. E. Setzler; Mr. Seymour Cohen; Mr. Nirmal S. Jain; Dr. Otto S. Kauder; Dr. Lawrence R. Brecker Mr. S. Gladstone; Dr. L. B. Nelson; Dr. Motonobu Minagawa; Mr. Leon Dubru Dr. Walter Weber; Mr. Alfred G. Williamson; Mr. S. C. Anand; Dr. Werner Mittlodorf

gd/GC,RB

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Exhibit # 43

Remarks By Dr. Lawrence R. Brecker

Presented At

OSH Hearing

On

VINYL CHLORIDE MONOMER

July 10, 1974

Argus Chemical Corporation is a wholly owned subsidiary of Witco Chemical Corporation, a public company listed on the N.Y. Stock Exchange.

We are a company of approximately 300 people with manufacturing plants in Taft, Louisiana and Brooklyn, New York that last year produced a total of 62 million pounds of chemicals valued at about $ 50,000,000.

The products we make can be broadly defined as polymer additives, that is chemicals that are used in small percentages of the recipes for converting various polymers to finished plastic products for use in industry, agriculture, and the home. Argus Chemical's, registered trademark products, DRAPEX© plasticizers, MARK © stabilizers, MARKURETA catalysts, and MARKSTAT© antistatic agents find use in a variety of polymers such as ABS polymers, cellulosics, polycarbonate, polypropylene, polyethylene, polystyrene, polyurethane, and polyvinyl chloride, (PVC for short). However, use in PVC accounts for 90% of the value of Argus products sold last year; certainly a shutdown of the PVC industry would force Argus to cease operators. Thus Argus and its employees are very much concerned that regulations intended to protect people from the hazards of vinyl chloride monomer (" monomer " for short) do so while still permitting the industry to produce the many useful products made of polyvinyl chloride.

The manufacture and sale of Argus products to the PVC fabricating industry results from laboratory activities carried out to formulate and test products for use in particular PVC recipes and consequently the need to formulate additives specifically for each intended use is well known and acknowledged in the Draft Environmental Impact Statement (DEIS) " Proposed Regulation Vinyl Chloride ", pages 39-42, 45-49.

Argus Chemical Corporation
subsidiary of Witco Chemical Corporation
633 Court Street, Brooklyn, New York 11231

Telephone 212—858-5678
Cable: Catchem, N.Y.

ARGUS CHEMICAL CORPORATION

Much of Argus' laboratory work involves compounding, processing and testing PVC on a small laboratory scale. Many representatives of each kind of additive listed on page 40 of the DEIS are used. The equipment and processes are laboratory replicas of those described at pages 42-44 of the DEIS, such as two-roll mills with 6 inch diameter rolls 10 inches wide and the Brabender Plasticorder which is a miniature Banbury type mixer and single screw extruder instrumented to measure melt viscosity as a function of temperature.

The scale of operation, however, in the plastics laboratory is completely different from PVC compounding or fabricating plants. A test batch of compound consists of 100 grams (less than 4 ounces) of PVC along with up to 60 grams of all other additives. The total quantity of PVC handled in a working day is typically 40 test batches, rarely as many as 50, for a maximum " throughput " of 11 pounds of PVC daily at Argus. While the trace monomer content of PVC varies between grades (and has been reported as high as 0.3% (3000 ppm)) we consider 0.01% (100 ppm) to be a conservatively realistic weighted average monomer content of the PVC used in our plastics laboratory. Thus, if one makes the quite improbable assumption that the <u>total amount of monomer</u> present in the PVC processed in one working day is released into the air of the workroom <u>without any removal</u> by ventilation, then the monomer concentration in the plastics laboratory at Argus can be calculated as 0.5 part per million. These calculations were made based on the dimensions of our laboratory 38 X 27½ X 12½ and assume no VCM removal by ventilation, which in fact is certainly not the case. It follows that for a laboratory scale operation with commercial grade PVC, the hazard that any standard is proposed to guard against simply does not exist.

We therefore submit that an exception of laboratory scale operations with PVC containing only trace quantities of monomer is reasonable and proper and respectfully solicit that the final standard when adopted, if not entirely exempting the fabricating of PVC products, at least be made only applicable to operations handling more than 100 pounds per working day..

Thank you.

2518

116

The B.F. Goodrich Company

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WILLIAM C. BECKER
ASSISTANT GENERAL COUNSEL
AND ASSISTANT SECRETARY

July 23, 1974

Mr. Julius Jimeno
Docket 36
1726 M Street, N.W.
Room 200
Washington, D.C.

Dear Mr. Jimeno:

As agreed to in Dr. Johnson's testimony, we submit the attached for the record.

Very truly yours,

William C. Becker

j
attach.

July 2, 1974

COMPILATION OF PRE-EMPLOYMENT LIVER FUNCTION TESTS AT ALL B.F.GOODRICH LOCATIONS FROM APPROXIMATELY MID-MARCH TO DATE

Each employee is identified by age, sex, race and the abnormal liver function test which precluded his employment.

Below is a summary of those rejected because of abnormal liver function tests:

Calvert City, Kentucky	4/23 or 17%
Long Beach, California	12/41 or 29%
Pedricktown, New Jersey	5/37 or 14%
Henry, Illinois	0/18 or 0%
Louisville, Kentucky	23/117 or 20%
Avon Lake, Ohio	34/175 or 19%

M. N. Johnson, M.D.
Director, Department of
Environmental Health

PRE-EMPLOYMENT BLOOD TESTS
CALVERT CITY, KENTUCKY

Age	Race	Sex	Observed Value/Normal Value
27	W	M	TB 1.8/1.5 - 1.3/1.0
36	W	M	GGTP 918457/28 (Tested three times)
31	W	M	LDH 161/155
27	W	M	LDH 215/155
			Normal
25	W	M	X
45	W	M	X
28	W	M	X
20	W	M	X
30	W	M	X
37	W	M	X
25	W	M	X
29	W	M	X
30	W	M	X
42	W	M	X
22	B	M	X
19	B	M	X
20	W	F	X
23	W	F	X
21	W	M	X
21	W	M	X
27	W	M	X
23	W	M	X
22	W	F	X
Total Normal			19
Total Other			4

PRE-EMPLOYMENT BLOOD TESTS
LONG BEACH, CALIFORNIA

(All Male)

Age	Race	Normal	Observed Value/Normal Value	
23	W		TB 1.1/1	LDH 123/120
18	B	X		
19	W	X		
18	W	X		
23	B		GGTP 42/28	
23	B		GGTP 92/28	
23	Sp. Am.	X		
21	Sp. Am.	X		
18	W	X		
23	B		GGTP 33.9/28	
21	W		LDH 126/120	SGOT 43/40
31	B		GGTP 65/28	
19	B		TB 1.1/1	
21	B		TB 1.1/1	
19	B		LDH 156/155	
21	W	X		
19	B	X		
18	B	X		
20	W	X		
20	W	X		
39	B	X		
35	Oriental	X		
20	B	X		
20	W	X		
24	W	X		
20	B	X		
21	W	X		
22	Sp. Am.	X	(Summer employee in Design Engineering)	
24	Oriental	X	(Summer employee in Lab)	
24	Oriental	X	(Summer employee in Lab)	
22	W	X		
22	Sp. Am.		LDH 197/155	SGOT 87/55
22	Sp. Am.	X		
26	B	X		
23	B	X	(Retested. Initial GGTP 30 - Final 20/28)	
18	B	X		
22	W		SGPT 96/53	SGOT 72/55
39	B		GGTP 35/28	
27	W	X		
20	W	X		
21	Am. Indian	X		

Total Normal 29

Total Other 12

PRE-EMPLOYMENT BLOOD TESTS
PEDRICKTOWN, NEW JERSEY

(All Male)

Age	Race	Normal	Observed Value/Normal Value	
21	W	X		
21	W		TB 1.6/1.5	SGPT 74/53
25	W		SGPT 106/53	
21	W	X		
29	W	X		
24	W	X		
23	W	X		
23	B	X		
40	W	X		
23	W	X		
20	B		SGPT 71/53	
26	B	X		
24	W	X		
24	W	X		
25	W	X		
18	W	X		
21	W		SGPT 158/53	SGOT 91/55
25	W	X		
28	W	X		
18	W	X		
19	W	X		
22	B	X		
20	W	X		
25	W	X		
20	W	X		
23	B	X		
21	W	X		
19	W	X		
28	W	X		
23	B	X		
27	W		SGOT 56/55	
19	W	X		
21	W	X		
20	B	X		
19	B	X		
23	W	X		
27	W	X		

Total Normal 32

Total Other 5

PRE-EMPLOYMENT BLOOD TESTS
HENRY, ILLINOIS

(All Male)

Age	Race	Normal
18	W	X
19	W	X
19	W	X
19	W	X
19	W	X
19	W	X
21	W	X
21	W	X
22	W	X
23	W	X
23	W	X
26	W	X
34	W	X
46	W	X
49	W	X
Total Normal		18
Total Other		0

PRE-EMPLOYMENT BLOOD TESTS
LOUISVILLE, KENTUCKY

Age	Race	Sex	Observed Value/Normal Value		
22	W	M	TB 2.2/1.0		
18	W	M	AP 117/85	SGOT 44/40	
19	W	M	AP 90/85		
41	W	M	AP 90/85		
18	W	M	AP 105/85	SGOT 50/40	
45	B	M	LDH 250/255	GGTP 47/28	
28	W	M	AP 127/85		
24	W	M	TB 1.1/1.0	AP 105/85	
22	W	M	TB 2.0/1.0		
25	W	M	TB 1.4/1.0		
25	W	M	TB 1.35/1.0		
20	W	M	TB 2.1/1.0		
22	W	M	TB 1.1/1.0		
28	B	M	TB 1.2/1.0		
32	W	M	TB 1.3/1.0	SGOT 50/40	
20	W	M	GGTP 32/28		
30	B	M	GGTP 29/28		
20	W	M	TB 1.8/1.0		
19	W	M	TB 1.2/1.0	AP 90/85	
51	W	F	AP 110/85	GGTP 36/28	
22	W	M	TB 1.4/1.0	SGOT 56/40	SGPT 65/25
20	B	M	TB 1.1/1.0	AP 90/85	
48	W	M	SGPT 34/25		
			Normal		
24	W	M	X		
21	W	M	X		
21	W	M	X		
18	W	M	X		
27	W	M	X		
32	W	M	X		
20	W	M	X		
25	W	M	X		
21	W	M	X		
21	W	M	X		
19	W	M	X		
18	W	M	X		
22	B	M	X		
22	W	M	X		
20	W	M	X		
26	W	M	X		
19	B	M	X		
21	W	M	X		
21	W	M	X		
24	W	M	X		

2523

PRE-EMPLOYMENT BLOOD TESTS
LOUISVILLE, KENTUCKY

Age	Race	Sex	Normal	Age	Race	Sex	Normal
19	W	M	X	20	B	M	X
19	B	M	X	36	W	M	X
25	W	M	X	23	W	M	X
23	W	M	X	20	W	M	X
18	W	M	X	20	W	M	X
22	W	M	X	27	W	M	X
41	W	M	X	26	W	M	X
19	W	M	X	21	W	M	X
22	W	M	X	19	W	M	X
20	W	M	X	27	B	M	X
20	W	M	X	19	W	M	X
26	W	M	X	27	W	M	X
22	W	M	X	46	W	M	X
20	W	M	X	43	W	F	X
20	W	M	X	50	W	F	X
30	W	M	X	34	W	M	X
25	W	M	X	21	W	M	X
24	W	M	X	20	W	M	X
23	B	M	X	18	W	M	X
18	W	M	X	21	W	M	X
23	W	M	X	21	W	M	X
30	W	M	X	24	W	M	X
20	B	M	X	21	W	M	X
27	W	M	X	19	W	M	X
19	W	M	X	30	W	M	X
28	W	M	X	19	W	M	X
28	W	M	X	40	W	M	X
22	W	M	X	45	W	F	X
27	W	M	X	21	W	M	X
40	W	M	X	23	W	M	X
18	W	M	X				
19	W	M	X				
20	W	M	X				
22	B	M	X				
24	W	M	X				
21	W	M	X				
29	W	M	X				
18	W	M	X				
18	W	M	X				
19	W	M	X				
21	W	M	X				
24	W	M	X				
23	B	M	X				
28	B	M	X				

Total Normal 94

Total Other 23

PRE-EMPLOYMENT BLOOD TESTS
AVON LAKE, OHIO - BOTH LOCATIONS

Age	Race	Sex	Observed Value/Normal Value		
19	B	M	SGOT 52/50	SGOT 61/50	LDH 229/200
19	B	M	(LDH 272/200	SGOT 50/50	GGTP 9.9/12.5
			(LDH 231/200	SGOT 73/50	TB 1.2/1.0
			(TB 1.5/1.5		
27	W	M	LDH 226/200	SGOT 82/50	AP 93/85
25	W	M	TB 1.1/1.0		
29	B	M	SGOT 55/50		
21	B	M	SGOT 124/50	TB 1.2/1.0	
20	W	M	SGOT 59/50		
23	B	M	GGTP 35/28		
27	W	M	SGPT 103/53	SGPT 73/53	SGPT 109/53
21	B	M	SGOT 61/55	SGPT 114/53	TB 1.0/1.5
23	B	M	GGTP 29/28		
23	W	M	(SGPT 123/53	SGPT 131/53	GGTP 37/28
			(SGOT 64/55		
25	B	M	SGPT 107/53	SGOT 77/55	SGPT 113/53
			SGOT 64/55		
19	B	M	SGOT 65/55		
20	B	M	TB 1.1/1.5		
20	W	M	LDH 220/200	SGOT 55/50	
23	W	M	SGPT 77/53	SGOT 71/55	GGTP 52/28
20	W	M	TB 2.1/2.5		
23	W	M	LDH 202/155		
20	B	M	SGPT 257/53	SGOT 172/55	GGTP 164/ 28
20	B	M	AP 228/148		
31	W	M	SGPT 53/53	GGTP 34/28	GGTP 29/28
19	W	M	TB 1.8/1.5		
23	B	M	SGPT 150/53	SGOT 109/55	GGTP 58/28
20	W	M	TB 1.6/1.5		
22	B	M	SGPT 107/53	SGOT 80/55	GGTP 81/28
20	W	M	SGPT 63/40		
20	W	M	SGPT 236/53	SGPT 157/53	
?	B	M	SGOT 34/50	TB 1.2/1.0	SGPT 114/53
?	B	M	SGOT 52/50	SGPT 87/53	
?	B	M	AP 93/85		
21	B	M	LDH 292/155		
24	W	M	TB 1.9/1.5		
20	W	M	(SGPT 93/53	GGTP 31/28	
			(SGPT 113/53	GGTP 45/28	

PRE-EMPLOYMENT BLOOD TESTS
AVON LAKE, OHIO - BOTH LOCATIONS

Age	Race	Sex	Normal	Age	Race	Sex	Normal
20	W	M	X	19	W	M	X
30	W	M	X	20	B	M	X
33	W	M	X	20	B	M	X
23	W	M	X	32	W	M	X
21	W	M	X	23	W	M	X
19	W	M	X	20	W	M	X
20	W	M	X	20	B	M	X
21	W	M	X	22	W	M	X
18	W	M	X	20	B	M	X
27	W	M	X	20	B	M	X
25	W	M	X	20	W	M	X
29	B	M	X	25	B	M	X
20	W	M	X	22	W	M	X
22	W	M	X	22	W	M	X
21	B	M	X	21	W	M	X
22	W	M	X	20	W	F	X
20	W	M	X	20	W	M	X
20	W	M	X	21	W	M	X
19	W	M	X	25	W	M	X
19	W	M	X	25	W	M	X
20	B	M	X	24	W	M	X
21	W	M	X	19	W	M	X
22	W	M	X	20	W	M	X
23	W	M	X	20	W	M	X
22	W	M	X	20	W	M	X
25	W	M	X	21	W	M	X
25	W	M	X	23	W	M	X
23	W	M	X	22	W	M	X
30	W	M	X	21	W	M	X
20	B	M	X	22	W	M	X
20	B	M	X	23	W	M	X
20	B	M	X	19	B	M	X
21	W	M	X	30	B	M	X
22	W	M	X	20	Sp. Am.	M	X
19	W	M	X	20	W	M	X
22	W	M	X	22	W	M	X
25	W	M	X	22	W	M	X
20	B	M	X	19	W	M	X
19	W	M	X	20	W	M	X
19	W	M	X	22	B	M	X
19	W	F	X	21	W	M	X
19	B	M	X	20	W	M	X
23	B	M	X	20	W	M	X
23	B	M	X	19	W	M	X

PRE-EMPLOYMENT BLOOD TESTS
AVON LAKE, OHIO - BOTH LOCATIONS

Age	Race	Sex	Normal	Age	Race	Sex	Normal
22	W	M	X	19	B	M	X
19	W	M	X	19	Sp. Am.	M	X
23	W	M	X	19	Sp. Am.	M	X
18	B	M	X	20	B	M	X
25	W	M	X	19	Sp. Am.	M	X
21	W	M	X	20	W	F	X
23	W	M	X	18	W	F	X
19	W	M	X	23	B	M	X
21	Sp. Am.	M	X	21	Sp. Am.	M	X
25	B.	M	X	24	W	M	X
22	Sp. Am.	M	X	29	W	M	X
24	B	M	X	22	W	M	X
19	W	M	X				
19	W	M	X				
19	W	M	X				
23	W	M	X				
22	W	M	X				
20	W	M	X				
21	W	M	X				
21	W	M	X				
19	W	M	X				
20	W	M	X				
19	W	M	X				
19	W	M	X				
19	W	M	X				
28	W	M	X				
20	B	M	X				
18	W	M	X				
34	W	M	X				
26	B	M	X				
19	B	M	X				
22	B	M	X				
18	B	M	X				
20	W	M	X				
18	W	M	X				
18	W	M	X				
21	W	M	X				
21	B	M	X				
20	B	M	X				
19	Sp. Am.	X	X				
18	B.	X	X				

Total Normal 141

Total Other 34

.25621

124

P.9 8/15

JOHN W. WHITTLESEY
COUNSELLOR AT LAW
270 PARK AVENUE
NEW YORK, N. Y. 10017
551-6797

REGISTERED MAIL
RETURN RECEIPT REQUESTED

August 12, 1974

Docket Officer
Docket OSH 36
Room 230
1726 M Street, N.W.
Washington, D.C. 20210

Re: Vinyl Chloride Presentation

Dear Sir:

It seems appropriate to comment for the record on behalf of Union Carbide Corporation, the employer of Vernon M. Jensen, President of Local 8-891 OCAW (AFL-CIO) who testified at OSHA's vinyl chloride hearings on July 10, 1974, as part of OCAW's overall presentation. Mr. Jensen is an employee of Union Carbide's Bound Brook, New Jersey plant, and head of the union representing our employees at that location.

The comments are made to put Mr. Jensen's testimony in perspective. It is clear that the material he presents therein is support, not for the "no detectable level" of exposure he advocates as a proper limitation on vinyl chloride, but in truth for the exemption of fabricating plants from the application of the standard entirely. The Bound Brook plant does not, it should be noted, produce polyvinyl chloride, but fabricates sheeting from the resin supplied from elsewhere; Mr. Jensen's comments have no bearing on the issue of what standard should govern polyvinyl chloride resin producers.

Mr. Jensen's testimony is that "exposure in the plant was probably particularly high in the plant from the 1940's through 1965... During those early years, we practically swam in polyvinyl chloride resin; so convinced were we of its harmlessness." This disturbed Mr. Jensen solely as a matter of hindsight, for nowhere did he allude to any recorded instance of angiosarcoma or any other deleterious effect on any employee from vinyl chloride exposure. The reason is clear; despite the alleged massive and lengthy exposures, no employees at the plant was affected thereby in any way, after review of the relevant records. This outcome clearly argues against,

not for, any need to curtail human exposure to vinyl chloride and for, not against, the exemption of the fabricating plants. Where there is no demonstrable hazard, in short, no protection is necessary.

Jensen's testimony does cite figures of occasional vinyl chloride concentrations that appear to be heavy. However, in the next breath, he states that no employee is actually exposed to such vinyl chloride monomer concentrations, which occur only in the van boxes (in which polyvinyl chloride resin is delivered to the plant), which he claims run up to 7000-9000 parts per million, and which our records show run merely to 2000-4500 parts per million. Such figures are under the circumstances hardly evidence of any employee hazard and can be disregarded as supporting a lower standard of exposure to vinyl chloride monomer than is presently required. Indeed, Jensen concedes that the employee who hooks up these same van boxes to the unloading airveying system has an exposure to vinyl chloride monomer of merely 2 parts per million. In this, he is accurate as to his facts; but here again, these facts clearly demonstrate that actual employe exposure, even where large vinyl chloride amounts are present in the vicinity of the work area, are small. The conclusion that should have been drawn by Jensen is that no company with similar circumstances to the above need establish regulated areas of more than minimal size to avoid vinyl chloride monomer exposure, and that dispersion of vinyl chloride into harmless concentrations in the atmosphere on release is rapid; and that the need for severe regulation is thereby vitiated.

Similar comments can be made on the Jensen testimony as to vinyl chloride concentrations in the plant's blender and preblending work areas where the resin is processed into sheeting. The concentrations of vinyl chloride anywhere outside the blending equipment itself is and long has been only 1 part per million (without controls) on the first floor of the building, and, on the mezzanine, occasionally up to (not an average of) 7 parts per million. The exposure of an operator to up to 175 parts per million while sampling is very occasional, not in any way frequent as Jensen implies, and even when it occurs, is for a period of less than a minute. It is clear that vinyl chloride, as a highly volatile gas, provides its own corrective to such excursions, the control of which is well under way at this time.

What Mr. Jensen has drawn, in short, is a picture of typical polyvinyl chloride fabricating operations which present no

hazard to the employees needing correction by the proposed OSHA vinyl chloride standard; of a management which, without OSHA prodding, has sought to maintain high standards of safety and health; of a plant, in summary, on which the proposed standard would impose wholly unnecessary cost requirements to meet limitations which it quite clearly does not need to meet. The Jensen testimony is a classic example of the reason why OSHA regulation of the sort proposed is plainly unwarranted.

Very truly yours,

JWW:me

John W. Whittlesey
Senior Labor Law Counsel
Union Carbide Corporation
270 Park Avenue
New York, New York 10017

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August 16, 1974

Mr. Jules Jimeno
Occupational Safety and
Health Administration
Docket OSH-36
Room 230
1726 M Street, N.W.
Washington, D. C. 20210

Dear Mr. Jimeno:

Enclosed are ten copies of a post-hearing memorandum submitted on behalf of Tenneco Chemicals, Inc., Air Products and Chemicals, Inc., and Hooker Chemical & Plastics Corporation. Please file this memorandum as a part of the record in the matter of a proposed permanent standard for occupational exposure to vinyl chloride.

Very truly yours,

Allan J. Topol
Counsel for
Tenneco Chemicals, Inc.,
Air Products and Chemicals, Inc.,
and Hooker Chemical & Plastics
Corporation

emf
Enclosures

bcc: George S. Flint, Esq. (w/enc)
Joseph T. Sebastianelli, Esq. (w/enc)
Mr. Raymond J. Abramowitz (w/enc)
J. T. Smith, Esq. (w/enc)

UNITED STATES DEPARTMENT OF LABOR

Occupational Safety and Health Administration

In the Matter of:
PROPOSED PERMANENT STANDARD FOR OCCUPATIONAL EXPOSURE TO VINYL CHLORIDE

POST-HEARING MEMORANDUM OF TENNECO CHEMICALS, INC., AIR PRODUCTS AND CHEMICALS, INC., AND HOOKER CHEMICAL & PLASTICS CORPORATION

Submitted by:

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Peter M. Phillipes
Theodore L. Garrett
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Counsel for Tenneco Chemicals, Inc., Air Products and Chemicals, Inc., and Hooker Chemical & Plastics Corporation

August 16, 1974

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UNITED STATES DEPARTMENT OF LABOR

Occupational Safety and Health Administration

In the Matter of:
PROPOSED PERMANENT STANDARD FOR OCCUPATIONAL EXPOSURE TO VINYL CHLORIDE

POST-HEARING MEMORANDUM OF
TENNECO CHEMICALS, INC.,
AIR PRODUCTS AND CHEMICALS, INC., AND
HOOKER CHEMICAL & PLASTICS CORPORATION

This memorandum is submitted on behalf of Tenneco Chemicals, Inc., Air Products and Chemicals, Inc., and Hooker Chemicals & Plastics Corporation. Tenneco is engaged in production of vinyl chloride monomer (hereinafter "VCM"), and polyvinyl chloride (hereinafter "PVC"), and in the processing of PVC into fabricated products. Air Products and Hooker are engaged in the production of PVC and the processing of PVC into fabricated products, but they do not produce VCM. Tenneco and Air Products testified at the hearing conducted by OSHA on June 25-28, and July 8-11. Hooker submitted a written statement of comments and objections for the record.

INTRODUCTION

In setting a permanent standard for occupational exposure to vinyl chloride, the Assistant Secretary of Labor

faces a complex decision with enormous potential consequence for the nation. He must recognize and weigh a variety of vital public interests including worker health and safety, economic and technological feasibility, and the likely domestic and international economic consequences of his determination. The importance of the Assistant Secretary's decision was discussed extensively at the rule-making hearing and the voluminous record that has accumulated bears witness to the major impact that decision will have on workers, industry and the general public.

In an effort to assist the Assistant Secretary in his deliberations, we have carefully reviewed the hearing record to determine what factual propositions are established by that record. Based upon this review, we submit that the record establishes the following five factual propositions:

(1) It is not technologically feasible for the PVC industry to meet the no-detectable level in the proposed OSHA standard. The proposed standard would require full-time use of respirators by all employees in monomer and polymer facilities for the foreseeable future. Such extensive use of respirators is neither feasible nor practicable and would actually be hazardous to employees.

(2) The lowest levels of exposure which even a majority of the PVC industry can feasibly meet are: (i) as

of October 5, 1974, a time weighted average (hereinafter "TWA") of 25 parts per million (hereinafter "ppm") and a ceiling of 40 ppm before donning of respiratory equipment is required; (ii) as of October 5, 1975, a ceiling of 25 ppm before respiratory equipment is required; and (iii) as of October 5, 1976, a TWA of 10 ppm with a ceiling of 25 ppm before respiratory equipment is required.

The lowest levels of exposure which even a majority of the VCM producing industry can feasibly achieve are: (i) as of October 5, 1974, a 10 ppm TWA and a 25 ppm ceiling before respiratory protection is required; and (ii) as of October 5, 1975, a 5 ppm TWA and a ceiling of 10 ppm before respiratory equipment is required.

(3) The proposed standard is economically infeasible under the court's decision in the Asbestos case.[1/] Discontinuance or substantial reduction of PVC production would have tremendously disruptive economic and social effects.

(4) The available scientific and medical evidence, i.e., human experience and animal data, does not establish that the exposure levels set forth in (2) above are unsafe levels. In fact, in the best available study of human experience, i.e., the Dow study, no adverse health effects were found at levels of exposure to VCM up to 200 ppm.

1/ Industrial Union Dept., AFL-CIO v. Hodgson, ___ F.2d ____, 1 OSHC 1631, No. 72-1713 (D.C. Cir., April 15, 1974).

(5) The proposed standard is not apposite to the situation of PVC processors and fabricators and would place unjustified operational and economic burdens on them, and therefore a separate and more appropriate standard should be developed to cover them.

Each of these five factual propositions will be discussed in detail below, with accompanying record references. At the outset we summarize the applicable legal requirement which OSHA must satisfy in promulgating the permanent standard.

The Occupational Safety and Health Act states that:

> "The Secretary . . . shall set the standard which most adequately assures, to the extent feasible, on the basis of the best available evidence, that no employee will suffer material impairment of health or functional capacity . . . ." 29 U.S.C. § 655(b)(5) (emphasis supplied).

This language was interpreted by the United States Court of Appeals for the District of Columbia Circuit in Industrial Union Dept., AFL-CIO v. Hodgson, ___ F.2d ____, 1 OSHC 1631, No. 72-1713 (D.C. Cir., April 15, 1974), to mean that OSHA cannot "protect employees by putting their employers out of business -- either by requiring protective devices unavailable under existing technology or by making financial viability generally impossible." ___ F.2d at ____, 1 OSHC at 1639. Thus the feasibility

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requirement of the statute has two aspects -- one technological and the other economic.[1/]

The technological aspect means that OSHA is not authorized to promulgate a standard which is not technologically feasible for the industry to achieve.[2/] The economic aspect means that the Assistant Secretary must weigh economic considerations in determining whether a standard is feasible. As the court stated in the Industrial Union Dept. case, "a standard that is prohibitively expensive is not 'feasible.'" ___ F.2d at ____, 1 OSHC at 1639.[3/] These economic considerations include many complex elements, such as impact on segments of the economy, and upon industry structure and international economic competition, as well as financial viability of individual, affected companies.

1/ This fact was recognized by OSHA. Boyd, Trans. p. 14.

2/ The court in the Industrial Union Dept. case did not determine precisely what percentage of an industry must be able to meet a standard before it becomes feasible. The court indicated on the one hand that a standard could be feasible even if one firm which lagged behind the rest of the industry could not meet that standard, and on the other hand that a standard would not be feasible if it could be achieved by "only a few leading firms." ___ F.2d at ____, 1 OSHC at 1639.

3/ In the course of the OSHA hearing, representatives of the Health Research Group attempted to downgrade the authority of this opinion, stating that a petition for rehearing had been filed with the Court of Appeals. (Cottine, Trans. pp. 430 (June 26, 1974), and 1553 (July 9, 1974)) The fact is, however, that on June 27, 1974, the Court of Appeals denied petitioners' petition for rehearing, and the petitioners in that case were advised by the Court that "it is not anticipated that any further action will be taken with respect to the suggestion for rehearing en banc." Letter from the Clerk, U.S. Court of Appeals for the District of Columbia Circuit, to George A. Cohen, attorney for petitioners, June 27, 1974.

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As will be shown, infra, the evidence establishes that the proposed standard for vinyl chloride is technologically infeasible, without regard to economic considerations. The technology simply does not exist to achieve the no-detectable level in the proposed standard. It will also be shown that if the proposed standard were promulgated, it would have drastic economic consequences.

In short, OSHA is not authorized to set a standard which the industry cannot meet by reason of technological or economic infeasibility. Adoption of a standard, such as the proposed permanent standard for vinyl chloride which is infeasible both technologically and economically, is therefore unauthorized by law and outside the scope of OSHA's authority.

THE FIVE FACTUAL PROPOSITIONS

With this background in mind as to the legal requirements consideration can be given to the five factual propositions which are established by the record.

1. The Proposed OSHA Standard of No-Detectable Level is Not Technologically Feasible.

The proposed standard, Section 1910.93q(f), is based upon the assumption that detectable levels of vinyl chloride can be eliminated through a combination of engineering

controls, work practice methods, and respirators. Extensive evidence was introduced at the hearing which decisively refutes this assumption. That evidence clearly establishes that it is not technologically feasible to reduce vinyl chloride exposure in either VCM or PVC plants to the no-detectable level by engineering controls and work practice methods. That evidence also establishes that the only means to prevent employee exposure to detectable amounts of vinyl chloride in monomer and polymer facilities would be to require full-time use of respirators by all personnel in the plant area. However, full-time use of respirators is neither a practicable nor a safe mode of operation. Therefore the likely consequence of a no-detectable exposure standard would be the shutdown of the PVC industry.[1/]

On this critical issue of whether it is technologically feasible to reduce exposures to the no-detectable level, the state of the evidence in the record is completely one sided. This evidence includes the testimony of Anton Vittone, President of B. F. Goodrich Chemical Company, who testified as chairman of SPI's committee of vinyl chloride monomer producers and PVC producers representing over 90

1/ See testimony of Anton Vittone (Goodrich-SPI), Trans. pp. 355-357; Dr. R. W. Strassburg (Goodrich-SPI), Trans. p. 581; Joseph Fath (Tenneco), Trans. pp. 687, 690-691; Harry E. Connors, Jr. (Diamond Shamrock), Trans. pp. 1030-1031; Statement by Raymond J. Abramowitz (Hooker), Exhibit 55, pp. 3-4.

percent of current U.S. capacity for VCM and PVC resin production.[1] Mr. Vittone testified that: "The Committee is unanimous in its position that the proposed permanent standard is not technologically feasible and, if adopted, would shut down the industry."[2]

This evidence also includes the testimony of engineers and operating personnel from both VCM and PVC manufacturing plants, who testified that the no-detectable level is not technologically feasible for their plants. Specifically, it includes the testimony of witnesses from Tenneco Chemicals, Inc. ("The standard of no detectable amount of ambient vinyl chloride is not feasible")[3]; Air Products and Chemicals, Inc. (requirements of the proposed standard are "impossible to meet")[4]; Dow Chemical Company

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1/ Vittone, Trans. p. 350.

2/ Vittone, Trans. p. 351. Mr. Vittone testified that the no-detectable level could not be achieved because it is not possible to eliminate in PVC production identifiable and "fugitive" losses of vinyl chloride from processing equipment such as valves, pumps, flanges and manheads, nor is it possible to dilute losses of vinyl chloride to the no-detectable level by ventilation. Trans. pp. 357-358.

3/ Fath, Trans. p. 686. A detailed exposition of the sources of vinyl chloride in PVC production and the factors which make it infeasible to eliminate these sources entirely was provided by Dr. Paul Lobo of Tenneco. See Trans. pp. 695-704.

4/ John G. Barr, Trans. p. 804. The testimony of Messrs. Richard Fleming and Barr contains a detailed description of the sources of VCM in PVC plants and the impossibility of eliminating these. See Trans. pp. 781-805.

("[I]t is technically infeasible to get to a no detectable limit of vinyl chloride monomer")[1]; Diamond Shamrock Chemical Company ("[T]he proposed 'no detectable' level is not feasible in the present state of the art or in the foreseeable future")[2]; B. F. Goodrich Chemical Company ("Achievement of no-detectable level of VCM exposure is not technically feasible")[3]; Uniroyal, Inc. ("At our PVC production facility it is not feasible to reduce airborne concentrations of vinyl chloride to zero")[4]; and Firestone Tire & Rubber Company ("[T]he exposure levels of the proposed standard are . . . technologically unachievable").[5]

1/ Oelfke, Trans. p. 901. Mr. Oelfke is responsible for coordination of vinyl chloride production techniques and technology for Dow Chemical on a worldwide basis. He discussed Dow's monomer production in detail, and he testified about the "problem areas" which have been identified and the number of valves, pumps and flanges required, which make complete elimination of leaks technically infeasible, even in a foreseeable new plant. Trans. p. 872.

2/ Connors, Trans. p. 1031. The basis for Diamond Shamrock's conclusion was described in detail by George D. Williams on the basis of his experience managing a PVC production facility. See Trans. pp. 1052-1059.

3/ Nelson, Trans. 1127. Mr. Nelson testified about the sources of VCM exposure in a PVC plant and the research and development work being conducted by Goodrich to reduce the exposure from these sources. Trans. pp. 1125-1127.

4/ Kleinfeld, Trans. p. 1492. The Kleinfeld testimony was accompanied by a report from Uniroyal's consulting engineers which provided a detailed analysis to support the Kleinfeld testimony.

5/ Walker, Trans. p. 1682. The Firestone testimony was accompanied by reports from outside consultants. See also the written statement of Stauffer Chemical Co.: "[T]he (cont'd.)

It is important to note that the testimony of industry witnesses as to the lack of feasibility was not based upon economic or cost considerations.1/ It was based solely upon technological infeasibility, i.e., we are not able to get to the no-detectable level in the present state of technology regardless of the dollars which are expended.

The importance of the testimony of these witnesses on the critical issue of technological feasibility cannot be minimized. They are experts, possessing the experience and knowledge of vinyl chloride and polyvinyl chloride production which is essential to an informed judgment on this issue. The testimony of these witnesses was accompanied by data and technical analyses. They were cross-examined at length, and all agreed to supply any additional data relating to technological feasibility which was requested. In the case of the Uniroyal and Firestone witnesses, their testimony as to

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(cont'd.) proposed standard is not feasible and will not be so within the foreseeable future"(p. 1); and the written statement of Raymond Abramowitz of Hooker Chemical & Plastics Corporation: "[I]t is the firm opinion of technical experts in our engineering and production departments that we could not continue to operate our plants and contemporaneously meet the proposed OSHA standard of 'no detectable level' of vinyl chloride" (p. 3).

1/ Vittone, Trans. pp. 417-418, 1180, 1186-1187; Fath, Trans. p. 747.

the lack of technological feasibility of the no-detectable level was corroborated by reports of outside consultants who studied their facilities.[1/]

The evidence shows that the proposed standard is infeasible not only in the case of existing PVC and VCM facilities, regardless of their age, but also in the case of facilities currently under construction and those that could be planned based upon "state of the art" technology though levels of exposure in new facilities will in all probability be lower than those of old facilities.[2/] Similarly, although it is easier to reduce levels of exposure in open-air plants in the southern United States than in closed facilities in the north, the no-detectable level is not feasible even in southern facilities.[3/]

With respect to PVC production the evidence in the record also includes detailed, reasoned analysis by Dr. Paul Lobo of Tenneco as to sources of vinyl chloride in such production and the factors which make it infeasible to eliminate these sources entirely. The PVC production process is a "batch" process which requires intermittent

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1/ Trans. pp. 1492, 1696-1699.

2/ Fath and Lobo (Tenneco), Trans. pp. 712-713; Roger Daniel (Dow), Trans. p. 892; Nelson (Goodrich), Trans. p. 1124.

3/ Lobo (Tenneco), Trans. p. 716; Dr. Langer (Dow), Trans. p. 1027; George Williams (Diamond Shamrock), Trans. p. 1068; Walker (Firestone), Trans. p. 1758.

opening of reactors. It has many complex origins for the release of VCM. These include the unloading of VCM delivered by truck or tank car; the need to open, inspect, and clean reactor vessels; the use of large volumes of air current in the drying stage; and finally the general unavailability of completely vapor tight pumps, valves and compressors.1/

Apart from these specific sources of emissions, in the operation of a PVC plant there are substantial losses of VCM which cannot be accounted for. The President of B. F. Goodrich Chemical Company testified that there was no feasible method to reach the no-detectable level because "losses from processing equipment will always occur. Some are identifiable and others are fugitive losses."2/ The production manager of a Diamond Shamrock PVC plant testified that engineering teams had "identified over 500 potential sources of vinyl chloride exposure," and that VCM emissions from these sources could not be reduced "to anywhere near an undetectable level."3/ In a post-hearing submission Dr. Lobo of Tenneco quantified theoretically the relationship between these fugitive emissions and VCM exposure in a plant.4/

1/ Lobo, Trans. pp. 697-702.

2/ Vittone, Trans. p. 357.

3/ Williams, Trans. p. 1053. See also Barr, Trans. p. 796.

4/ See Exhibit B to Tenneco submission of August 15, 1974.

With respect to VCM monomer production there is comparable testimony from representatives of Dow Chemical Company as to the nature of such production and the sources of VCM exposure for employees. Mr. Oelfke described problem areas and the impossibility of eliminating leaks from the large number of valves, pumps and flanges involved.[1/]

In contrast to the extensive evidence establishing the technological infeasibility of the proposed standard, none of the participants at the hearing introduced any substantial evidence to show that the proposed standard can be achieved technologically.[2/] The testimony which attempted to point in this direction came from representatives of the National Institute of Occupational Safety and Health (hereinafter "NIOSH"), union representatives, representatives of the American Chemical Society and Calgon Corporation. Taken individually or collectively none of this testimony constitutes substantial evidence that the no-detectable level is feasible.

The contribution of NIOSH on technological feasibility consisted of a table (Exhibit 13, Table 2, NIOSH submission) showing sources of emissions of vinyl chloride

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1/ Oelfke, Trans. pp. 871-872.

2/ The Department of Labor admitted that the proposed permanent standard was developed by OSHA without the benefit of engineering, technological, or economic studies of a formal nature. (Trans. pp. 1110, 1112)

and suggesting work practices which could be used to reduce employee exposure.1/ However, on cross-examination the NIOSH witnesses admitted that NIOSH had made no technological or economic feasibility studies and that NIOSH's suggestions were based only upon a "walk-through" inspection of several facilities.2/ More important, the NIOSH witnesses testified that they did not get the impression from their examination of industrial facilities that a no-detectable level of vinyl chloride could be achieved, and they did not even have "any idea" as to what the exposures would be if the suggested work practices were instituted.3/

The union representatives who testified that the proposed standard was technologically feasible likewise admitted that their conclusions were not based on any in-depth study of the problem. For example, Anthony Mazzocchi of the Oil, Chemical and Atomic Workers International Union testified that a no-detectable standard was feasible by October 1974, but Mr. Mazzocchi conceded that this conclusion was not based upon any technological or engineering studies.4/

Similarly, testimony by representatives of the American Chemical Society (ACS) appeared to state that the

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1/ See Trans. pp. 104-105.

2/ Trans. pp. 104-105, 125, 133.

3/ Trans. pp. 131, 137.

4/ Trans. p. 1442.

proposed standard was technologically feasible if industry were provided with a sufficiently long period of time to allow for the detailed engineering design and development of equipment necessary for full compliance with the standard.[1/] The representatives of the ACS conceded that this conclusion was not based upon any technical studies or plant surveys, or even upon the views of the membership of the ACS as a whole. Instead it was based upon the opinions of individuals on an ACS committee whose backgrounds and experience were not disclosed, and who were not available for cross-examination.[2/] One member of this ACS committee, who is employed in industry, has stated in a post-hearing submission that he never reviewed and he "cannot support the final position taken by ACS."[3/]

Finally, Calgon Corporation, a marketer of activated carbon, testified that beds of activated carbon could be used to remove VCM from air or nitrogen streams.[4/] Serious

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1/ Trans. pp. 1323, 1355.

2/ Trans. pp. 1339, 1343, 1357-1359, 1365-1366.

3/ See letter from Zeb G. Bell, Jr., to John Stender, July 31, 1974, p. 3.

4/ Trans. pp. 1881-1885. This simplistic hypothesis is well known to the chemical community, i.e., that organic chemical vapors such as VCM are adsorbed by carbon to a greater or lesser extent. See, e.g., Kirk-Othmer, Encyclopedia of Chemical Technology (1964), p. 149.

doubts, however, have been raised as to the effectiveness of Calgon's activated carbon technology for any purpose related to the removal of VCM. Calgon representatives admitted that use of activated carbon for the recovery of VCM was an "embryonic science"; that Calgon had no expertise or experience in installing activated carbon beds in facilities such as vinyl chloride monomer and polymer plants; that Calgon's projections of efficacy were based upon laboratory experiments using small quantities of carbon and VCM, and small air streams at ambient temperature; and that the VCM used in their experiments was unaccompanied by other chemicals as would be the case in plant-scale operations.1/ Calgon representatives admitted that their experiments did not yield any definitive results about the likelihood and potential scope of polymerization on the surface of an activated carbon bed in actual plant operations.2/ Mr. Lovett from Calgon even candidly stated that "whether it [activated carbon] can be applied to a vinyl chloride plant or not has not been demonstrated."3/

In this regard Calgon had no engineering or technical proposals as to how their carbon might be used in the

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1/ Trans. pp. 1885, 1930, 1934-1935.

2/ Trans. pp. 1965-1967.

3/ Trans. p. 1945.

removal of VCM from the ambient air in a PVC or VCM manufacturing plant. Thus even if Calgon's activated carbon could effectively remove VCM from an air stream, the evidence clearly shows that its use would be primarily directed to the reduction of emissions to the atmosphere, and not to solving the problem of in-plant worker exposure to VCM.1/

More important, Tenneco is the PVC manufacturer, referred to in the Calgon testimony, which has experimented with activated carbon in a pilot plant operation.2/ Tenneco has filed a post-hearing submission describing its laboratory and pilot plant testing of activated carbon and the data accumulated to date.3/ The conclusion reached by Tenneco from these tests is that the use of activated carbon is not feasible to achieve no-detectable or even low levels of vinyl chloride in manufacturing plants.

In sum, there is no substantial evidence from which it could be concluded that the proposed permanent

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1/ Trans. pp. 1953-1955.

2/ See Exhibit A to Tenneco submission of August 15, 1974.

3/ Id. at 2-4. See also Lobo, Trans. p. 700.

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standard is technologically feasible. All of the evidence in the record establishes that the no-detectable level in the proposed standard could not be achieved by engineering controls and work practice methods. Therefore the only means to prevent employee exposure to detectable amounts of vinyl chloride in monomer and polymer facilities would be to require full-time use of respirators by all personnel in the plant area. However, the evidence establishes that full-time use of respirators is not an acceptable, safe or practicable means of meeting the proposed standard.

Indeed, there was near unanimity among government, industry and labor witnesses that full-time use of respirators would not be an acceptable solution to the problem of worker exposure to vinyl chloride. OSHA's own witness, Dr. Hyatt, stated with regard to respirators "At best, any of these devices are [sic] an instrument of torture."[1/] Other consultants and experts were unanimous in their opinion that the full-time use of any type of respirator is unsafe and infeasible.[2/]

Expert testimony on this issue was fully supported by experienced operating personnel from several companies.

1/ Trans. p. 92/14.

2/ See Dr. J. F. Tomashefski, Trans. pp. 312-316; Robert D. Soule, Trans. pp. 573-579; Strassburg, Trans. p. 581.

Philip Scarito of Tenneco stated that, on the basis of 18 years experience as a PVC plant manager, use of respirators by PVC workers 100 percent of the time they spent in regulated work areas would be "neither an appropriate, safe nor feasible solution to the problem of worker exposure to vinyl chloride monomer since it would entail substantial health and safety risks in and of itself."[1/] Mr. Scarito then pointed out that, even with occasional rather than constant use of respirators, Tenneco already had suffered one lost time accident when a worker became entangled in an air hose attached to a breathing apparatus and fell from a ladder.[2/]

Among the health and safety factors cited by expert witnesses and operating personnel were: restriction of vertical and horizontal vision; the possible hazard caused by an air supply either being depleted or contaminated; possible interference with hearing preventing a worker from noticing audible alarms; fatigue, discomfort and claustrophobia; interference with verbal communications; the difficulties and dangers of moving around a vinyl chloride or PVC facility trailing a long air hose; exacerbation of worker respiratory illness, especially by respirators

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1/ Trans. p. 705.

2/ Trans. p. 705. See also Statement of Raymond Abramowitz (Hooker), Exhibit 55, p. 11.

used in the pressure demand mode; and the risk of a false sense of protection where there exists an inadequate fit.1/

Witnesses representing organized labor expressed skepticism about use of respirators full-time as a solution. For instance, Mr. Jensen of the Oil, Chemical and Atomic Workers International Union stated "even a normal healthy person would have difficulty breathing over a long period of time with a respirator, particularly an effective one."2/

OSHA itself appears well aware of the problems involved in the full-time use of respirators, and this awareness is reflected in the proposed permanent standard. Section 1910.93q(g)(2) states that: "Respirators shall be used only in cases of emergency . . .. Respirators may not be used in lieu of feasible engineering controls or work practice methods." However, as demonstrated above, there do not exist technological and engineering methods to reduce the VCM level to the no-detectable level in the proposed standard. Therefore, promulgation of the proposed

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1/ See Hyatt, Trans. pp. 88-90, 92/7-9, 92/12; Tomashefski, Trans. pp. 306-316; Soule, Trans. pp. 577-579; Strassburg, Trans. pp. 583-584, 629-630; Philip Scarito, Trans. pp. 705-708; Oelfke, Trans. p. 897; Williams, Trans. pp. 1054-1056; Francis F. Hoy, Trans. pp. 1711-1713. See also Comments and Objections of Stauffer Chemical Company, p. 12.

2/ Trans. p. 1432.

standard necessarily will bring about a result -- full-time use of respirators -- which the authors of the proposed standard recognize as neither acceptable nor feasible.[1/]

2. The Society of Plastics Industry's Proposed Levels For VCM and PVC Producers are the Lowest Levels Which Even a Majority of the Industry can Feasibly Achieve.

For the reasons indicated in Section 1 above, the no-detectable level in the proposed standard is not feasible. The next factual determination which must be made is: What is the lowest level of exposure which can be feasibly achieved by the industry utilizing a combination of engineering controls, work practices and the wearing of respirators for periods which are not excessive. This determination must be made on the basis of a preponderance of the evidence in the record.

When all of the evidence is reviewed, there is only one conclusion which can be reached. The levels of exposure proposed by Anton Vittone (President of B. F. Goodrich Chemical Company), as an SPI witness, represent

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1/ Industry witnesses recognized that respirators can be used and must be used in the case of excursions above certain specified levels. However, the levels of exposure proposed by industry witnesses presume a tolerable maximum use of respirators. See, e.g., Vittone, Trans. p. 360 ("The utilization of respiratory equipment for this purpose would not be excessive, and, therefore, feasible"). See also Lobo, Trans. p. 704.

the lowest levels of exposure which even a majority of the industry can feasibily achieve.

Mr. Vittone described the following standard for PVC plants "as representing the maximum reduction which a majority of the industry believes feasible": (i) as of October 5, 1974, a TWA of 25 ppm and a ceiling of 40 ppm before donning of respiratory equipment is required; (ii) as of October 5, 1975, a ceiling of 25 ppm before respiratory equipment is required; and (iii) as of October 5, 1976, a TWA of 10 ppm with a ceiling of 25 ppm before respiratory equipment is required. (Trans. pp. 358-359)

Mr. Vittone described the following standard for VCM plants as representing "the maximum reduction which a majority of the industry believes feasible": (i) as of October 5, 1974, a 10 ppm TWA and a 25 ppm ceiling before respiratory protection is required; and (ii) as of October 5, 1975, a 5 ppm TWA and a ceiling of 10 ppm before respiratory equipment is required. (Trans. p. 361)[1/]

Mr. Vittone testified that the SPI proposal was developed on the basis of "a judgmental consideration of the members of the industry" with every company cognizant of the available technology and their own data. (Trans.

1/ A lower level is proposed for monomer production because monomer production, unlike PVC production, is a closed process.

pp. 380-381) The SPI proposal was endorsed by a majority of the industry, although not all companies agreed that they could meet the SPI proposed levels. (Vittone, Trans. p. 380)

The evidence establishes that the SPI proposed levels are the lowest levels which even a majority of the industry can achieve. These levels "represent ambitious, difficult levels of operational exposure dependent on large expenditures of money, reduced capacity, and the development of technology not currently in hand."1/ They are the lowest levels which can be feasibly achieved in view of technological limitations, entirely apart from any "economic considerations."2/

The SPI proposed levels presuppose the use of respirators in the event of excursions above the proposed ceilings. Thus respirator use would be maintained at a tolerable level for employees.3/

There is evidence that the standard is too restrictive in describing the types of respirators which can

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1/ Vittone, Trans. p. 361.

2/ Vittone, Trans. pp. 417-418, 1180, 1186-1187; Fath, Trans. p. 747.

3/ Vittone, Trans. p. 360.

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be used, and that while no type of respirator can be used full-time, to the extent that respirators must be used in the case of excursions under the SPI proposal, half rather than full-face respirators should be permitted as well as canister or air-purifying devices for limited use.[1/]

The two-year phasing down to a TWA of 10 ppm in PVC facilities and 5 ppm in VCM facilities is based upon estimates of the time required to investigate and to order and install the new equipment required to get to these levels.[2/] Typical lead times for purchase and installation of plant equipment run approximately two years.[3/] It can also be expected that delays beyond normal lead time will be encountered because the entire industry will be purchasing similar equipment at the same time.[4/] Thus the two-year lead time is the absolute minimum that could conceivably be regarded as feasible.

1/ See Hyatt, Trans. pp. 92/11-12; Tomashefski, Trans. p. 316; Soule, Trans. pp. 576, 580; Strassburg, Trans. pp. 585-587 (Reports on extensive experiments done on canisters by B. F. Goodrich); Oelfke, Trans. p. 895; V. K. Rowe (Dow), Trans. p. 972; Hoy, Trans. pp. 1745-1748; Statement of Raymond Abramowitz (Hooker), Exhibit 55, pp. 10-11.

2/ Vittone, Trans. pp. 353-354; Fath, Trans. pp. 692-693, 723-724.

3/ Lobo, Trans. pp. 702-703; Barr, Trans. p. 801. See also Exhibits D-1 and D-2 of Tenneco submission of August 15, 1974, consisting of recent articles describing lead times required for the purchase of various industrial items.

4/ Fath, Trans. p. 693; Kleinfeld, Trans. p. 1532.

A number of witnesses from PVC producers testified that their companies expected to achieve the SPI proposed level of 10 ppm TWA by October 5, 1976, for PVC plants, but that the reduction to 10 ppm represents the maximum feasible reduction in worker exposure. Representatives from Tenneco, for example, testified that "[i]t will be feasible" for Tenneco to achieve the levels proposed by SPI,[1/] and that these represent the "lowest technologically feasible levels"[2/]; representatives from Diamond Shamrock testified "we believe we will be able to comply with the exposure levels recommended earlier in these hearings by SPI"[3/]; and representatives from B. F. Goodrich testified "we endorse the SPI proposal on the stepwise reduction of vinyl chloride levels in PVC work areas."[4/] The testimony of these companies was based upon data as to their prior exposure levels, the particular circumstances of their plants, and their experience with PVC production and knowledge of available technology. The witnesses were experts, and they were subject to cross-examination.

1/ Fath, Trans. p. 694.

2/ Fath, Trans. p. 747.

3/ Connors, Trans. p. 1031.

4/ Nelson, Trans. p. 1127.

There is comparable testimony by witnesses from Dow Chemical Company, a monomer producer, that Dow expects to achieve the SPI proposed level of 5 ppm TWA as of October 5, 1975, but that reductions below this level are not feasible.1/ This Dow conclusion was based upon the complexity of its production plant, what it has done in the past, and the problems it faces in the future. Similarly, Tenneco, which produces vinyl chloride monomer as well as PVC, also supports the SPI proposed levels for VCM production.2/

There is no credible evidence that it would be technologically feasible for the PVC industry to achieve levels below 10 ppm TWA or that it would be feasible for the VCM industry to achieve levels below 5 ppm TWA in the foreseeable future.

On the contrary, witnesses from some PVC producers testified that the SPI proposed levels represent projections which may not be achievable. For example, representatives of Air Products testified that

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1/ Oelfke, Trans. pp. 899-903.

2/ Fath, Trans. pp. 688, 691.

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though Air Products believed the October 5, 1974, standard of 25 ppm TWA proposed by SPI to be feasible, it would be difficult and expensive to attain and to meet continuously; and the lower levels proposed by SPI were "much more difficult and much more speculative as to achievement at this time."[1/] Firestone representatives testified that "Firestone's engineering studies indicate that it is impossible on the basis of the present state of technology and the engineering art to predict with any degree of certainty that the reduced levels suggested by SPI can be achieved in existing facilities."[2/] Similarly, Firestone's consultant engineer, Dr. Higgins, stated that extensive and costly new ventilating and exhausting systems would be required if 10 ppm TWA was the level required by the standard, but that "no definite forecasts can be made as to whether this low level [10 ppm TWA] can be attained."[3/] In a written submission Hooker Chemical & Plastics Corporation stated that: "We are not yet certain that these [the SPI] levels are technically attainable, but we nevertheless consider these to be good goals."[4/] Thus, even if the SPI proposed levels are

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1/ Fleming, Trans. pp. 789-790.

2/ Walker, Trans. pp. 1684-1685.

3/ Walker, Trans. pp. 1705-1706.

4/ Exhibit 55, pp. 7-8.

incorporated in the permanent standard, some companies will find it necessary to make applications for variances.1/

As indicated above, industry witnesses testified in detail about the levels of VCM exposure which they believe to be attainable in VCM and PVC plants. Much of this testimony also included a description of the type of monitoring to be employed in determining vinyl chloride exposure because it is not possible to separate levels of exposure from the method by which these levels are determined.

On this point there is extensive testimony that the personnel sampling method in the proposed standard gives an imperfect and incomplete picture of the plant's dynamic environment.2/ Even if the personnel monitoring samples are taken at representative times, they will inevitably miss peak levels of exposure due to temporary excursions, which are of primary concern in accurately recording and limiting employee exposure to vinyl chloride. Thus OSHA may have accumulated data indicating that many

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1/ "Temporary variances may be obtained when timely compliance is technologically impossible." Industrial Union Dept., AFL-CIO v. Hodgson, ___ F.2d at ____, 1 OSHC at 1639, n. 23.

2/ Fath, Trans. p. 719. In addition, it should be noted that there may be doubt as to the accuracy of monitoring carried out by OSHA field compliance inspectors. See statement of P. A. Ketterer, Exhibit C to Tenneco submission of August 15, 1974.

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readings in plants are currently below the 25 ppm proposed by SPI as a TWA for the October 5, 1974, standard. However, the constant sequential area monitoring system, proposed by industry witnesses gives a more complete and accurate picture of actual levels.1/

The record also establishes that constant sequential area monitoring will have other advantages. It will allow prompt warning to workers so that they can don respiratory protection when excursions above the ceiling occur.2/ Area monitoring enables immediate action to correct hazardous situations because it provides information on present concentrations rather than information about the past day's concentrations.3/ This means that if a leak occurs in the plant, personnel monitoring will not alert employees to the fact that a leak has occurred until the results of the personnel monitoring tests are available. In contrast, area monitoring will permit prompt detection and correction of the leak.

1/ Vittone, Trans. p. 359; Barr, Trans. p. 799.

2/ Vittone, Trans. pp. 359, 384-385, 434-437; Fath, Trans. pp. 719-722; Walker, Trans. p. 1722.

3/ R. H. Stehl (Dow-SPI), Trans. pp. 556-557; Rowe, Trans. p. 972; Walker, Trans. p. 1722.

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As a result of these factors, Joseph Fath of Tenneco testified that constant sequential area monitoring "will do more to prevent and correct prolonged dosages of vinyl chloride than any other mechanical correction possible in our existing plants."[1/] Mr. Fath also testified that such a system will enable more rapid and reliable diagnosis of the existence of problem areas, and will yield a permanent record of exposures throughout the plant on a complete and constant basis.[2/]

Despite the necessity for and benefits of constant sequential area monitoring, testimony establishes that the integrity of the constant sequential area monitoring system should be checked by periodic use of personnel monitoring.[3/]

In summation, if the decision on this critical issue is to be made on the basis of the evidence in the record, as it must be, then the proposed standard should not contain any levels of exposure lower than those in the SPI proposal. All of the evidence establishes that these represent the lowest levels which even a majority of the industry can achieve. There is no

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1/ Trans. p. 692.

2/ Trans. p. 692.

3/ Stehl, Trans. pp. 568, 593; Fath, Trans. p. 738; Barr, Trans. p. 799; Nelson, Trans. p. 1127.

substantial evidence to support a conclusion that the attainment of any lower levels is technologically feasible.

3. The Proposed Standard is Economically Infeasible. A Shutdown or Serious Disruption of the PVC Industry Would have Drastic Effects on the U.S. Economy.

In the Industrial Union Dept. case the court held that economic considerations, including macroeconomic consequences, of a standard are appropriate criteria by which to judge its feasibility. It was amply demonstrated in testimony at the hearing on the vinyl chloride standard that PVC is a major contributor to the American economy. Its withdrawal from the market would have serious adverse economic consequences.

Representatives of Arthur D. Little, Inc., testified that an immediate shutdown of the PVC industry could result in the loss of 1.7 to 2.2 million jobs and a loss of domestic production of $65 to $90 billion annually.1/

Total PVC production in 1973, of approximately five billion pounds, was the second largest volume of any plastic.2/ PVC is regarded as the nation's most versatile thermoplastic polymer.3/ It has important end-use

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1/ Trans. p. 456.

2/ Ralph L. Harding, Jr. (SPI), Trans. pp. 330-331.

3/ Fath, Trans. p. 690.

applications in building and construction, the motor vehicle industry,[1] apparel and home furnishings, phonograph records and communications equipment and in a variety of other miscellaneous products ranging from credit cards to medical tubing. As a consequence there are thousands of companies involved in the processing of PVC or the fabricating of vinyl plastic products.[2] Certain highlights of the testimony regarding PVC's economic importance are as follows:

> Ten percent of PVC production is used in production of insulated wire and cable. PVC insulated wire can be extruded rapidly at low cost, is flame resistant, has high electrical characteristics, and as a consequence is the most widely used wire in ship and building construction, communications equipment and automobiles. If PVC were to become unavailable it would take three to five years to replace PVC in the wire and cable industry. This time would be needed to develop substitute compounds

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1/ In a statement submitted for the record on July 15, 1974, General Motors Corporation stated that a disruption in the supply of PVC could seriously upset its production capability, affecting 450,000 GMC employees.

2/ Trans. pp. 458-459.

and to undertake retooling and plant redesign.[1/]

PVC materials are used extensively in preparing space launch vehicles for NASA programs and in the construction and overhaul of nuclear submarines.[2/]

Twenty-five percent of PVC is used in the manufacture of pipe. Sixty percent of the pipe manufactured by one major manufacturer, Johns-Manville, uses PVC; appropriate substitutes are currently unknown and their availability is dubious even if they were found; extensive retooling would probably be required before pipe could be manufactured from substitute compounds.[3/]

1/ Arthur D. Little, Inc., Trans. pp. 458-459; William Nuckols (General Cable Corporation), Trans. pp. 1601, 1608 (If PVC insulated cable were not available "most construction in this country would come to a halt").

2/ Trans. pp. 1535-1545.

3/ Trans. pp. 1210-1211.

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PVC is unexcelled as a long-life sealant for application in airfield and bridge construction. No substance has been found that even approaches PVC with regard to performance and economic feasibility as a sealant.1/

PVC is essential to the U.S. footwear industry if it is going to continue to compete with foreign manufacturers. No available substitute is known. Failure of PVC supply, affecting the shoe industry would have harsh consequences for many small towns and rural areas since 80 percent of the footwear manufactured in this country is produced in towns with populations under 20,000.2/

Another potential economic consequence of OSHA's proposed permanent standard would be the take-over of U.S.

1/ Posh Chemical, Inc., Trans. pp. 1859-1860; Superior Products, Trans. p. 1877.

2/ American Footwear Industries Ass'n., Trans. pp. 1639-1640, 1642-1643.

markets by foreign competitors, less stringently regulated.[1/] Also there is the prospect that many small fabricators would not be able to afford the costs of retooling for use of substitute materials even if such became available. The consequence would be the closing of numerous small businesses.[2/]

The foregoing facts clearly establish that the proposed standard, and any other standard more onerous than that advanced by the SPI witnesses, would not be "feasible" within the meaning of the governing statute as interpreted in the Industrial Union Dept. case.

4. The Available Scientific and Medical Evidence Does Not Establish That the Levels Proposed by SPI are Unsafe.

Extensive medical and scientific testimony, including human experience and animal data, was presented on the relationship between vinyl chloride and angiosarcoma of the liver -- a form of liver cancer. That testimony does not establish that the levels proposed by SPI are unsafe, but rather affirmatively suggests the contrary.

A number of medical and scientific witnesses testified that at sufficiently high levels for sufficiently long periods of time, exposure to vinyl chloride could

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1/ Walker, Trans. p. 1695; W. L. Schmir (Fields Plastics & Chemicals), Trans. pp. 1838-1839.

2/ Kleinfeld, Trans. p. 1495.

cause angiosarcoma.[1] However, these witnesses further testified that there is a "dose response relationship"[2] between exposure to vinyl chloride and angiosarcoma, that there might be a safe or threshold level of exposure below which vinyl chloride does not cause angiosarcoma, and that this threshold level has not yet been determined.[3] Thus this testimony does not establish that the levels proposed by SPI are unsafe levels.[4]

The most significant evidence relating to the issue of whether the SPI proposed levels are safe is the

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1/ See generally the testimony of Drs. Kraybill (Trans. pp. 24-31, 49-81), Schneiderman (Trans. pp. 32-81), Key and other NIOSH representatives (Trans. pp. 93-137), Selikoff (Trans. pp. 212-285), and Keplinger (Trans. pp. 438-453).

2/ Kraybill, Trans. p. 68.

3/ Dr. Selikoff testified on cross-examination that we cannot say whether exposures to low levels of vinyl chloride are safe or unsafe. (Trans. p. 268) See also the testimony of Dr. Key, Trans. pp. 94-95, 129-130. Dr. Keplinger testified that: "There is a level where you just don't get it [a carcinogenic response]," Trans. pp. 448-449. Dr. Schneiderman, when asked on cross-examination whether there are safe levels for chemical carcinogens, testified: "[W]e don't know." Trans. p. 60. Dr. Kraybill and Dr. Schneiderman testified that a large-scale government research project has been undertaken to determine if there is a safe level for chemical carcinogens. (Trans. pp. 63-64) Dr. Gehring testified that: "Where exactly that threshold lies for man, obviously has not been determined." (Trans. p. 995) See also the statement of the Assistant Secretary relating to the standard for 14 carcinogens. 39 Fed. Reg. at 3758 (1974)

4/ Dr. Key testified that even if a chemical is carcinogenic at very low levels of exposure, at those low levels the latency period might be extended beyond the life expectancy of humans and "an acceptable level" can be justified. (Trans. p. 123)

data of Dow Chemical Company, which was the subject of the testimony of Drs. Cook and Holder of Dow.[1/] The Dow data is particularly significant because it is the only evidence in the record which relates actual levels of exposure of employees to the health of those employees. Dow was able to conduct this study because Dow is the only company in the industry which has determined levels of vinyl chloride exposure for many years. Thus Dow could relate the results of medical examinations of each of its employees to the levels of vinyl chloride to which that employee was exposed. The conclusion reached by Drs. Holder and Cook is that there was an increased incidence of medical abnormalities, including malignancies, in workers exposed to levels above 200 ppm of vinyl chloride; but workers exposed to levels of vinyl chloride less than 200 ppm did not have an increased incidence in any medical abnormality or in malignancies.[2/] Dr. Holder concluded that: "Our data would indicate that below 200 ppm there is no adverse effect."[3/] Dr. Rowe, the Director of Toxicological Affairs in Health and Environmental Research for Dow, reviewed this

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1/ Trans. pp. 932-955.

2/ Cook, Trans. pp. 933, 937, 938; Holder, Trans. pp. 941, 948-949, 954, 955.

3/ Trans. p. 980.

data and also concluded that with exposures up to 200 ppm there is "no evidence of any adverse effect on any organ system attributable to vinyl chloride" and there have not been observed "any medical abnormalities attributable to vinyl chloride."1/

Dr. Gehring, also of Dow, supplied a possible explanation, based upon tests he had conducted, as to why low levels of exposure might not have any adverse effect upon humans while higher levels might produce angiosarcoma.2/ Dr. Gehring testified that the ultimate carcinogen is very likely not vinyl chloride itself but a substance produced by the metabolism of vinyl chloride in the body.3/ Dr. Gehring's data showed that vinyl chloride "may be metabolized via different pathways depending upon the magnitude of exposure."4/ There is a primary or normal pathway by which vinyl chloride may be metabolized at low levels and that pathway may not produce a carcinogen. Once that primary pathway becomes saturated, which occurs at a sufficiently high level of vinyl chloride exposure, then the vinyl chloride may be metabolized by an alternate pathway which produces

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1/ Rowe, Trans. p. 973.

2/ Gehring, Trans. pp. 955-970.

3/ Gehring, Trans. p. 958.

4/ Rowe, Trans. p. 971.

the ultimate carcinogen. Thus at high exposures the metabolism of vinyl chloride may lead to the production of a carcinogen, but at lower exposures no carcinogen may be produced in the body.[1/]

Extensive evidence was presented about the 13 cases of angiosarcoma which had occurred among employees of PVC plants in the United States. All of this evidence is consistent with the proposition that the levels of exposure proposed by SPI are not unsafe levels. The record establishes that there are 36 vinyl chloride plants in the United States, and that all 13 cases occurred among employees of four plants. Seven of these 13 cases occurred among employees of one plant; three among employees of another plant; two among employees of a third plant; and one among employees of a fourth plant.[2/] All four of these plants are older than 20 years. The levels of vinyl chloride exposure which prevailed during the 1940's and 1950's in the industry and at the plants where the angiosarcoma cases have occurred are not known with precision. Therefore no definitive conclusion can be drawn as to exact levels of exposure to which these 13 employees were exposed.

1/ See Gehring, Trans. p. 969.

2/ Key, Trans. p. 97.

However, all of the testimony established that levels of exposure during the 1940's and 1950's were substantially higher than levels that prevail in the industry today.[1/] One method used to seek approximation of historical levels was to poll workers regarding their memory of the presence of the odor of VCM, and though there is dispute about the odor threshold for VCM, such a polling revealed that levels substantially greater than 250 ppm and in some cases substantially greater than 1,000 ppm were common prior to 1960.[2/] Dow introduced actual monitoring data gathered during the 1950's showing TWA's as high as 385 ppm, and excursions up to 4,000 ppm.[3/]

Thus it seems apparent that all cases of angiosarcoma of the liver discovered to date among PVC workers

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1/ See, for example, the testimony of Dr. Kraybill that NIOSH data indicates that levels may have ranged from 1,000 to 2,000 ppm and higher. (Trans. p. 29) Dr. Nicholson testified that some employees could have been exposed to levels in excess of 1,000 ppm. (Trans. p. 272) Mr. Vittone testified: "[W]e believe that the average exposure in the decade of the forties could have been some forty times greater than they are today. With respect to peak exposures, they no doubt were some hundred times of today's emergency standard of 50 parts per million . . .." (Trans. pp. 352-353. See also Fath, Trans. pp. 689-690.

2/ C. U. Dernehl (Union Carbide), Trans. pp. 510-511; Fleming, Trans. pp. 786-787; J. E. Ertel (Robintech), Trans. pp. 504-505, 525-526.

3/ Daniel, Trans. pp. 888-889.

occurred as a result of exposures substantially greater (by an estimated factor of 40) than those proposed by SPI.[1/] This evidence thus affirmatively indicates that the levels of exposure proposed by SPI are not unsafe.

Other evidence which points to this same conclusion relates to the fact that there are a total of eight PVC plants in the United States which are more than 20 years old.[2/] This means that among the eight plants older than 20 years, seven angiosarcoma cases occurred at one plant; three at another; two at another; one at a fourth; and no cases occurred among employees at four plants.[3/] One possible explanation for this non-random distribution of angiosarcoma cases among the plants older than 20 years is that exposure levels may have been higher at the four plants which had cases than at the other four plants, and that the levels of vinyl chloride exposure in the four plants older than 20 years which had no angiosarcoma cases may have been below the threshold needed to cause angiosarcoma.[4/]

1/ Vittone, Trans. pp. 352-353; Fleming, Trans. pp. 786-787.

2/ In view of the latency period for angiosarcoma, which might be approximately 20 years, consideration in this analysis is given only to plants older than 20 years in an effort to avoid the objection that the plant has not been in operation long enough to have any cases.

3/ Key, Trans. p. 97.

4/ Kraybill, Trans. pp. 55-56. Dr. Key testified that "job duties and degree and length of exposure" may account for

(cont'd)

There was also extensive testimony about the results of animal experiments conducted by Professor Maltoni and by the Industrial Bio-Test Laboratories. These inhalation studies on mice and rats produced angiosarcomas of the liver in at least some experimental animals at various levels of exposure down to 50 ppm.[1/] Dr. Keplinger, who is directing the study at Bio-Test testified that the Bio-Test results are "preliminary in nature" and that the study "is still in progress."[2/] Dr. Keplinger also testified that "one can not say that an average PPM of 25 is unsafe because the studies have never been done."[3/]

(cont'd) the fact that the cases occurred only in these four plants and not in others. (Trans. p. 97) Dr. Lloyd testified that the level of exposure at each plant would affect the distribution of angiosarcoma cases among plants. (Trans. pp. 111-113) Dr. Peters testified that the dose and the level of exposure could account for the fact that some plants had angiosarcoma cases and others did not, and that if the levels of exposure were lower at the plants which had no cases, this "would certainly suggest" that the levels in these plants were safe levels. (Trans. pp. 766-767) Dr. Selikoff testified that the risk of cancer was reduced with lower exposures. (Trans. pp. 268-269) Dr. Kraybill also testified that there may have been another agent present, which enhances the development of angiosarcoma, and without the presence of this agent the angiosarcoma would not have developed. (Trans. pp. 30-31, 56-57)

1/ See Maltoni Telegram, Trans. pp. 11-12; Kraybill, Trans. pp. 26-28, 61-62; Keplinger, Trans. pp. 439-443.

2/ Keplinger, Trans. pp. 439-440, 443.

3/ Keplinger, Trans. p. 443. Dr. Kraybill testified that any opinions as to whether angiosarcomas would be induced in animals at levels below 50 ppm represented "speculation." (Trans. p. 62)

Quite apart from the preliminary nature of the results, the testimony raised substantial questions about the validity of extrapolating animal results to humans. Dr. Selikoff testified that you cannot "automatically translate animal data to human beings."[1/] Dr. Kotin testified that "I just don't think the animal data are that informative."[2/] Dr. Rowe of Dow testified that we do not really know what significance to attach to the dose response data of the animals and we need to know much more before determining which animal species is most like man in its metabolism of VCM.[3/] Dr. Harris of Uniroyal testified that the animal results to date do no more than indicate that a problem exists.[4/] Dr. Marvin Kuschner, in a report appended to the Firestone submission, stated that it is enormously difficult to extrapolate between species, and that it is proper to inquire into the possibility that man is less susceptible than rodents to the effects of vinyl chloride.[5/] Dr. Michael Baden, a Firestone witness, stated

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1/ Trans. p. 260.

2/ Trans. p. 834.

3/ Trans. p. 971.

4/ Trans. pp. 1307-1308.

5/ Trans. pp. 1686-1687. Dr. Keplinger testified that the mouse is more susceptible than the other two species of animals tested. (Trans. p. 443)

that "[o]ne can extrapolate only with great care and difficulty from any animal species" and that when human evidence is available "that is what should be looked into and evaluated in setting standards up."[1/] This point was well summarized by Dr. Keplinger, who is conducting the animal experimentation. Dr. Keplinger testified that "[t]he relevance of these data in mice for prediction or extrapolation to effects on man of the same concentration, same durations of exposure, are quite conjectural."[2/]

Dr. Schneiderman of NCI discussed certain means to extrapolate mathematically from the rodent data to derive acceptable risk levels for humans, such as the probability of finding one angiosarcoma case among 100,000 workers. The extrapolation is a two-step process: (i) extrapolating from animal experiments at moderate or high doses to predict animal responses at very low exposures[3/]; and (ii) translating the results of this extrapolation into risk levels for man.[4/] For the first step Dr. Schneiderman derived levels that were extremely low -- less than a quarter of a part per million exposure -- to produce a risk of one angiosarcoma in 100,000 animals.[5/] He testified

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1/ Trans. p. 1768.

2/ Trans. p. 443.

3/ Schneiderman, Trans. p. 34. This prediction in theory yields a probability of finding one angiosarcoma among 100,000 animals at the very low exposures.

4/ Trans. p. 44.

5/ Trans. p. 42.

that the levels of exposure which would produce this same result in humans would even be lower.[1/]

There is extensive evidence which shows the invalidity of Dr. Schneiderman's statistical projections. This is the evidence, summarized on pages 43-44, supra, which establishes the speculative nature of translating the results of animal experiments to man. There is also the evidence presented by Dr. Gehring of Dow which challenges the extrapolation made by Dr. Schneiderman from results obtained on animals at high levels of exposure to predict results at low levels of exposure for these same animals. On the basis of the data from his actual experimentation, Dr. Gehring challenged Dr. Schneiderman's basic assumption -- that the metabolic behavior at exposures above a threshold is the same as that below.[2/] If the behavior differs, and Dr. Gehring showed by data that it does in the case of vinyl chloride, then extrapolation is invalid. It is not scientifically sound to use data based upon levels of exposure exceeding thresholds to predict what "will occur at lower concentrations or lower exposures."[3/]

1/ Trans. p. 45.

2/ Gehring, Trans. pp. 958-959.

3/ Gehring, Trans. pp. 969-970. Further testimony on the concept of thresholds was presented by Dr. Paul Kotin, formerly director of the National Institute of Environmental Health Sciences at NIH, who testified with witnesses for (cont'd.)

The epidemiological and toxicological evidence which is outlined above cannot lead to the conclusion that the levels of exposure proposed by SPI are unsafe. Indeed, based upon all of the evidence in the record, and particularly the Dow data, the levels proposed by SPI appear to be levels which are safe for employees. Dr. Gehring of Dow stated that in his opinion, based on toxicological and human data, there is no indication that 50 ppm "constitutes an unreasonable risk."1/

The debate between those who believe that extrapolations can or cannot be made from animals to man and that so-called safe thresholds of human exposure to a carcinogenic substance can or cannot be established is inconclusive

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(cont'd.) Air Products. According to Dr. Kotin, there is no threshold for a carcinogen in terms of the interaction of a carcinogen with a single molecule of a cell. However, there is a threshold for producing a cancer in a total organism. Dr. Kotin testified: "[Y]ou have to have two things to have a clinical cancer: the carcinogen, and then you must have a series of other influences which take this transformed cell -- which is going on in our bodies all the time -- and move it along the road to a clinical cancer." (Trans. pp. 831-833)

1/ Trans. pp. 1023-1024. See also the statement of Dr. J. Wister Meigs of Yale Medical School included as Appendix C to the Comments and Objections of Stauffer Chemical Co. Stauffer endorses levels equivalent to the proposed SPI levels and Dr. Meigs opines "based upon the available medical evidence and human experience data, it is my opinion that the worker exposure levels proposed by Stauffer are reasonable." (p. 6)

and continuing. Certainly there is no evidence in the record to establish that the levels of exposure proposed by SPI are unsafe.

5. The Proposed Standard is Not Apposite to the Situation of PVC Processors and Fabricators and Would Place Unjustified Operational and Economic Burdens on Them, and Therefore a Separate and More Appropriate Standard Should be Developed to Cover Them.

The evidence establishes that employees in PVC processing and fabricating facilities are exposed to very low levels of vinyl chloride at the present time. Dr. Key of NIOSH testified that a survey of fabrication facilities disclosed breathing zone levels of VCM ranging between 0.3 ppm and 12 ppm, with an estimated TWA of 2 ppm.[1/] A panel of witnesses appearing for SPI testified and submitted extensive data showing that exposures in fabricating plants are generally less than 1 ppm, except at mixing, unloading and storage areas, and that increased ventilation in these areas can reduce monomer levels below 1 ppm.[2/] In addition

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1/ Trans. pp. 105-106.

2/ Paul Beebe, Jr. (Goodyear), Trans. pp. 636, 368-639 (survey of five plants); Arthur A. Smith (Robintech), Trans. pp. 642-643; Rodney P. Becker (Diamond Shamrock), Trans. pp. 648-651; Philip J. Weaver (Goodrich), Trans. pp. 653, 655-660, 668. See also Fleming, Trans. p. 794; and Statement of Louis Molinini (Gollob Analytical Service Corp.), Exhibit 20-0 (survey of 20 plants).

there is evidence that as to these areas, existing OSHA regulations relating to air contaminants provide for adequate precautions.[1/]

The evidence also establishes that, quite apart from levels of exposure, the administrative provisions of the proposed standard would impose a serious burden upon the PVC processing and fabricating industry which is carried out by at least 3,000 companies, many of which are small businesses.[2/] These administrative provisions relate to such things as monitoring, medical surveillance, protective clothing, controlling entry and exit from areas, and posting signs and labels. Rimar Manufacturing, Inc., for example, testified that "an all encompassing regulation such as this which is proposed . . . would preclude the possibility of a small businessman being allowed to participate in any way within the polyvinyl chloride industry."[3/] Alco Standard Corporation testified that the requirements for respirators, protective clothing, and medical surveillance would be so costly that serious thought would have to be given to dropping its PVC processing business.[4/]

1/ Fleming, Trans. p. 794. The regulations are included in 29 C.F.R. § 1910.93.

2/ Trans. pp. 653-654.

3/ Robert I. Martin, Trans. p. 1241.

4/ O. B. Hager, Trans. p. 1471. The requirements for controlling entry and exit, washing and monitoring would cause costly and severe disruption to processor activities. Armstrong Cork Company, Trans. p. 1251.

All of this evidence points to the conclusion that processors and fabricators should not be covered by the permanent standard currently under consideration. The Assistant Secretary should, therefore, issue, in proposed form, a separate and appropriate standard for this industry and he should allow an opportunity for public comment in response to the proposal. Additional information could then be submitted on the relationship between residual monomer content in resin and worker exposure in processing and fabricating facilities with a view toward exempting processors and fabricators from any standard if they employ only resin with a residual monomer content below a level which would be specified in the processor and fabricator standard. This relationship between residual monomer content and employee exposure in processing and fabricating facilities was the subject of testimony at the hearing. The majority of the industry proposed a goal of reducing residual monomer content to 100 ppm by October 5, 1977, and thereby substantially eliminating employee exposure in processing and fabricating plants.1/ On the other hand,

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1/ Vittone, Trans. p. 363; Fath, Trans. pp. 693-694 (Tenneco believes that reduction of residual monomer levels is the best way to assure absolute minimum exposure to the 99 percent of the PVC worker population who are employed in PVC processing and fabricating plants).

Air Products testified that 500 ppm residual monomer content represents a sufficiently low level to prevent residual monomer from posing any problem for fabricators.[1/] Diamond Shamrock introduced data to support the conclusion that residual monomer content need not be reduced below 1,000 ppm.[2/] It is apparent that OSHA does not have enough data yet to draw precise conclusions about the relationship between worker exposure in processing and fabricating facilities and residual monomer content in resins. This is, however, an area that deserves further and extensive examination.[3/]

This proposal of dealing equitably and appropriately with processors and fabricators by issuing a separate proposed standard is not barred by the statutory mandate to the Secretary to issue a permanent standard within six months after promulgation of an emergency temporary standard, because the emergency temporary standard relating to vinyl chloride does not apply unequivocably or unambiguously to processors and fabricators.[4/]

1/ Fleming, Trans. pp. 793-795; Barr, Trans. pp. 802-803.

2/ Becker, Trans. p. 651; Connors, Trans. p. 1032.

3/ The standard for processors and fabricators could also include provisions for ventilation and monitoring which are appropriate as well as others which are relevant to this industry.

4/ The emergency temporary standard states on the one hand: "This section applies to any area or operation in which (cont'd.)

In summary, the proposed standard, which was the subject of the hearing, would work serious hardship on numerous small businesses whose problems of compliance and whose levels of worker exposure differ radically from those of VCM and PVC producers. OSHA should develop a separate and more appropriate standard for this industry.

CONCLUSION

The evidence in the record supports each of the five factual propositions discussed in this memorandum. First, the proposed permanent standard of no-detectable level of vinyl chloride is not technologically feasible, and if promulgated in its current form would be unauthorized and unlawful. Second, the levels of exposure proposed by SPI represent the lowest levels of exposure which even a majority of the industry can feasibly achieve. Third, the proposed standard is economically infeasible and would have tremendously disruptive economic consequences. Fourth, the available medical and scientific evidence affirmatively indicates that the levels of exposure proposed by SPI are

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(cont'd.) vinyl chloride . . . is manufactured, reacted, handled, processed, released, repacked, or stored." 39 Fed. Reg. 12343, April 5, 1974. On the other hand it states: "This section does not apply to the handling, storage, or other use of vinyl chloride polymers and copolymers in the form of fabricated products." Id.

not unsafe. Fifth, the proposed standard would place unjustified burdens on PVC processors and fabricators, and a separate and more appropriate standard should be developed to cover them.

Respectfully submitted,

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and Hooker Chemical & Plastics
Corporation

August 16, 1974

Arthur D Little, Inc.

130

August 19, 1974

TO: Gordon J. Myatt
Administrative Law Judge

FROM: Vince Ficcaglia

SUBJECT: Submission of Arthur D. Little Inc., Report
Department of Labor Hearings on Vinyl Chloride
Occupational Exposure Standard
Post-Hearing Submission

Enclosed please find three (3) copies of an Arthur D. Little, Inc. report entitled "Polyvinyl Chloride, The U.S. Industry: An Impact Analysis". The report is being submitted as part of the post-hearing materials as agreed to during our testimony before you on Wednesday, June 26, 1974.

VC:br
Enc.

130

Polyvinyl Chloride

The U.S. Industry: An Impact Analysis

Arthur D Little, Inc.

2553

UNITED STATES POLYVINYL CHLORIDE

INDUSTRY IMPACT ANALYSIS

A Report to

The Vinyl Chloride and Polyvinyl Chloride Producers Committee

The Society of The Plastics Industry

August 1974

Arthur D Little Inc

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I. SUMMARY

The recent discovery of a relatively high concentration of angiosarcoma among workers in a few polyvinyl chloride (PVC) resin plants has led the Occupational Safety and Health Administration (OSHA) to issue emergency exposure standards. More recently, OSHA has proposed to replace the emergency temporary standard of 50 parts per million (ppm) with a permanent acceptable tolerance level which requires that vinyl chloride monomer (VCM) be "non detectable" in regulated (i.e., work) areas.

The companies in the PVC resin industry have stated that they would be unable to meet the newly proposed "non detectable" tolerance standard and its enforcement would force the shutdown of all PVC resin plants in the U.S. Such a shutdown would have a significant adverse impact on production and employment levels and would not be restricted solely to the VCM and PVC industries, but would permeate throughout many sectors of the U.S. economy.

In an attempt to identify the extent and quantify the magnitude of the potential impact of such a shutdown, The Society of The Plastics Industry (SPI) commissioned Arthur D. Little, Inc. (ADL) to undertake an analysis to identify and estimate the loss in employment and domestic production that would be generated by such an event. It was not the purpose of this study to assess whether the proposed permanent "non detectable" standard of OSHA would indeed force an immediate shutdown of all PVC resin plants. At no point in this study did ADL assess the probability of such a shutdown.

Summarizing the conclusions of this report, we find that an immediate shutdown of all PVC resin plants in the U.S. and the subsequent unavailability of PVC resin could result in a loss of 1.7-2.2 million jobs in consuming and related industries and a loss of domestic production value of $65-90 billion annually. Because of the known widespread use of PVC

resins in the U.S., many sectors of the economy would be affected, with the greatest impact occuring in the building and construction and automotive areas. The extent to which materials could be substituted for PVC resin in the immediate term (less than one year) is quite limited and, therefore, would have an insignificant effect upon reducing the potential job and domestic production losses noted above. In addition, the worldwide tightness in supplies of PVC resin precludes the possibility of reducing the above impacts through increased imports of this material.

II. INTRODUCTION

The recent discovery of a relatively high concentration of angiosarcoma, a rare form of liver cancer, among workers in a few polyvinyl chloride (PVC) resin plants has led the Occupational Safety and Health Administration (OSHA) of the U.S. Department of Labor to issue emergency exposure standards. In its emergency pronouncement, OSHA set a safety tolerance standard of 50 parts per million (ppm) for the vinyl chloride monomer (VCM) content in the atmosphere of the plant work areas. For many of the PVC industry's participants, such a tolerance standard was close to the norm already being experienced and enforced in their plants. This lower level of VCM exposure is well below the initial maximum of 500 ppm set by OSHA when it promulgated its original safety standards for the plastics industry.

More recently, OSHA has proposed to replace the emergency temporary standard of 50 ppm with a permanent acceptable tolerance level which requires that VCM be "non detectable" in regulated (i.e. work) areas. The OSHA proposal as announced would require that the VCM tolerance standard be lowered to 0-1 ppm by October 1974. This standard is in agreement with a recommendation already put forth by the National Institute of Occupational Safety and Health (NIOSH).

The companies in the PVC resin industry have stated that they would be unable to meet the newly proposed "non detectable" tolerance standard announced by OSHA. The industry has concluded that adoption of the proposed permanent standard as currently written would force the shutdown of all PVC resin plants in the U.S. Such a shutdown would have a significant impact on industry and commercial activities, resulting in substantial losses of production and jobs. These losses would not be restricted solely to the VCM and PVC industries but would permeate throughout many sectors of the U.S. economy dependent upon the myriad of products based upon PVC resins.

In an attempt to identify the extent and quantify the magnitude of the potential impact of such a shutdown on the U.S. economy, The Society of The Plastics Industry (SPI) commissioned Arthur D. Little, Inc. (ADL) to undertake an analysis to identify and estimate the loss in employment and domestic production that would be generated by such an event.

III. INDUSTRY IMPACT ANALYSIS

A. Overview of Methodology

The purpose and scope of our task was to identify and estimate the potential economic impact resulting from a complete and immediate shutdown of all PVC resin plants in the U.S.* The impact has been expressed in terms of the potential effect on current domestic production and employment in the U.S. economy. Because of the known widespread use of PVC resins in numerous applications in the U.S. today, the potential for lost production and employment would permeate beyond just the PVC resin, VCM, and their immediate customer markets. Because of the high degree of interdependence among sectors of the economy and lack of adequate substitute materials, production cutbacks in PVC-related markets would lead to further output reductions in a whole host of materials and service supplying industries.

In undertaking our analysis, we focused upon measuring the magnitude of the economic impact resulting from an immediate shutdown of all PVC resin plants in the U.S. With this as our basic focus, our approach included the following:

1. Identification of PVC resin usage by product and end-use application: to best identify the use of PVC resin in the U.S. economy, we defined five end-use applications modules which include:

    a. Building and Construction
    b. Motor Vehicles
    c. Home Furnishings, Household Goods and Apparel
    d. Specialty Products
    e. Miscellaneous Products

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* It was not the purpose of this study to assess whether the proposed permanent "non detectable" standard of OSHA would indeed force an immediate shutdown of all PVC resin plants. At no point in this study did ADL assess the probability of such a shutdown.

Construction of these modules allowed us to identify in a clear and complete fashion the flow and use of PVC resin in the U.S. today. Figures A-1 through A-5 included in Appendix A identify the product and end-use applications of each module.

2. Estimation of resulting economic impact from shutdown of all PVC resin plants: in order to estimate the potential production and job loss from such an event requires an analytical framework that is a balanced, internally consistent and current model of the U.S. economy which identifies the PVC resin industry and defines its relationship to other intermediate and end-use markets. Input/Output econometric models have this capability and ADL uses this type of model in its economic forecasting and regional impact analysis work. The estimates presented in this report are based upon the current relationships that exist within the ADL Input/Output Model. Originally based upon the Input/Output table developed by the U.S. Department of Commerce, Office of Business Economics for 1963, the model has since been revised, expanded, and updated by ADL staff members.

3. Assessment of the extent of PVC resin dependency in each module: in order to accurately estimate the potential impact resulting from an immediate shutdown of PVC resin plants, it was necessary to make explicit assessments of the extent of PVC resin dependency in each end-use application. This estimation was largely necessitated by our use of published Federal Government data which is classified according to Standard Industrial Classification (SIC) definitions.* Since the data available on the intermediate and end-use markets of each module included both vinyl and non-vinyl related products, estimates were made of the proportion that is dependent upon PVC resin. Our analysis at this stage

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* Standard Industrial Classification Manual, Executive Office of the President, Office of Management and Budget, 1967.

indicated that the dependency upon PVC resin varies significantly among the end-use applications. For example, dependency is extremely high in such end-use applications as phonograph records, communications and building wiring, and medical products. On the other hand, PVC resin is not a primary material in paint manufacture.

4. Assessment of the potential for immediate substitution of other raw materials for PVC resin: the resulting economic impact based upon the analysis undertaken in Steps 1-3 had to be reassessed to incorporate the potential for immediate substituion of other raw materials for PVC resin. In an effort to develop our best estimates of this potential, we gathered a team of ADL staff members with considerable experience in the areas of plastics and related markets and the product sectors which a priori offer themselves as possible substitution candidates. We supplemented our own experience with field interviews of selected fabricators and purchasers of PVC-based products.

In summary, the ADL team concluded that because of the unique nature of PVC resin, it would not be possible to duplicate the exact properties of vinyl plastic products by using substitute raw materials. Even if it were possible, the costs of production and the resulting selling prices of the final products would probably be prohibitive in many cases. Furthermore, it would still take up to an additional three to four years to complete all of the necessary research and development leading to the required changes in product, process, and equipment design, and the actual delivery and installation of new equipment in order to make non-vinyl equivalent products available on a commercial scale necessary to satisfy the demands of industry.

The findings of our analyses undertaken in Steps 3 and 4 above are discussed at greater length in Chapter IV. The following sections of this chapter present a brief overview of the PVC resin industry, background information on I/O models, and a review of the economic impact results.

B. The PVC Resin Industry

1. Introduction

Vinyl-type plastics are among the oldest of the major plastic materials. The first commercial plant to make PVC resin, the basic material for vinyl plastic products, was constructed in 1939. Over time, the vinyl plastics industry was developed largely by the major rubber and chemical companies. The initial vinyl plastics were rather similar to rubber products in that they were very flexible or "rubbery" in nature. Because of this, companies that processed or fabricated rubber compounds into such forms as sheets and tubes (or hose) could use their existing equipment (e.g., calenders and extruders) to process vinyl as well as rubber compounds. Today, about two-thirds of vinyl plastic products are still rubbery (or elastomeric) in nature, but now PVC is also made into rigid end products such as pipe, siding and phonograph records. There are currently 21 separate producers of PVC resin operating 36 plants in the U.S.

PVC resin is a powdery material which is made from vinyl chloride monomer (VCM--a gas under standard conditions of temperature and pressure and a liquid when transported and stored under pressure). PVC resin, as such, cannot be processed but must be made into a vinyl compound which contains several other additives (such as plasticizer, stabilizer, and colorant) in addition to the basic resin, in order to fabricate it into useful product forms. Because they can be compounded to yield a wide range of materials with specific properties, PVC resins are used in more applications than any other type of plastic material.

2. Vinyl Chloride Monomer (VCM)

The VCM industry is made up of nine producers. Four companies (Shell, Dow, PPG, and Allied Chemical) produce VCM for merchant sale only and together they account for over half of the current total VCM capacity. The other five producers make VCM largely or completely for captive use in making PVC resin.

Last year the production of VCM was about 5.3 billion lbs valued at about $292 million (about 5.5¢/lb on average). This year VCM production will likely approximate 5.5 billion lbs but at about 9¢/lb; the 1974 VCM market value will be nearly $500 million. This production required roughly 2.8 billion lbs of ethylene and 3.7 billion lbs of chlorine as raw materials having a combined market value of about $300 million. Total industry capacity is now about 6.4 billion lbs of VCM which should only be sufficient to supply the U.S. demands for about two more years. This assumes that exports which were about 420 million lbs last year, or 8% of domestic production, are decreased substantially. At present, no new VCM capacity is under construction in the United States. Since it takes approximately 2 1/2-3 years to build and start up a VCM plant. decisions on new capacity expansions are needed in the very near future to allow for PVC resin growth beyond 1976. Last year about 88% of all VCM production was used domestically to make PVC resin; another 4% was used domestically for other applications and the remaining 8% was exported. (See Table I).

3. PVC Resin

The term PVC resin is used to describe a family of synthetic polymers in which the vinyl chloride monomer content is over 50% by weight. In practice, the VCM content is usually 85% or more. Resins which are made from VCM only are called homopolymers and those containing other monomers (such as vinyl acetate) are called copolymers. Homopolymers represent about 85% of total PVC resin output.

The total production of PVC resin last year was about 4.5 billion lbs valued at about $650 million (14.5¢/lb). This year production will likely be about 4.6 billion lbs valued at $920 million (20¢/lb, on average). About one-quarter of the total world production of PVC resin is made in the United States. The industry rated capacity of the 21 existing PVC resin producers is now about 5 billion lbs/yr. However, at least one-third of the existing resin producers are expanding their capacity and at least two new producers are entering the market. By the end of 1975 the total PVC resin capacity will

TABLE I

PRODUCTION, PRICE, VALUE, PRODUCERS AND USES OF VINYL CHLORIDE MONOMER

Production, Price, and Market Value

Year	Production (billion lbs)	Average Price (¢/lb)	Market Value ($ million)
1962	1.3	7.5	98
1967	2.4	5.3	127
1972	5.2	4.2	218
1973	5.3	5.5	292
1974	5.5	9.0	495

Producers and Capacity

Producer	Annual Rated Capacity (million lbs) (at August, 1974)
Shell	1,500
Dow	1,200
Goodrich	1,000
PPG	800
Continental Oil	625
Ethyl	420
Monochem (Borden and Uniroyal)	350
Allied Chemical	300
American Chemical (Stauffer and Atlantic Richfield)	170
Total	6,365

End-Use Pattern - 1973

Use	Approximate Percent of Market
PVC Resin	88
Export and Other	12
Total	100

Sources: U.S. Tariff Commission and Arthur D. Little, Inc., estimates.

be about 6.4 billion lbs. This should be sufficient to serve the U.S. market through 1978 (see Table II).

PVC is the most versatile type of synthetic resin produced and is used in more individual end products than any other type of plastic material. It is possible to identify over 25 individual end use product areas for PVC, each of which accounts for between 1 and 29% of the total PVC resin market. By grouping these products into broad market categories it is possible to estimate that over half of all PVC resin is used to make products that are classified as building and construction materials. This includes pipe, conduit, flooring, wire and cable insulation, siding, panels, window frames, wall covering, trim, and gaskets and sealing materials. Home furnishings and household goods taken together represent about 12% of the overall PVC resin usage. This broad grouping of products includes furniture upholstery and decorative laminates (using PVC printed films) for furniture and TV cabinets, table covers and shower curtains, garden hose, appliance components (e.g., hoses and gasketing) and window shades. The end-use market called consumer goods, which also represents roughly 12% of PVC resin use contains items such as phonograph records, footwear (e.g., shoes, boots), toys, dolls and games, apparel and other outerwear, sporting and athletic goods, luggage, handbags and many other products. Packaging materials, such as flexible and rigid film (used for packing fresh and processed meats, produce, cookies and candy as well as non-food products--e.g., blister packs), bottles, and can and closure lining (coating) materials account for about 8% of total PVC resin sales. The transportation equipment industry (mainly motor vehicles) in total represents about 6% of the PVC resin market. The major products include upholstery materials, trim, headliners, wire and cable insulation, floor mats, and external (e.g., landau-type) tops.

The remaining 9% of the domestic PVC resin market is composed of a miscellaneous products category containing hundreds of individual applications. Among the most significant are: medical products (e.g., tubing, blood bags), stationery supplies, book covers and bindings, conveyor belting, credit cards, tools and hardware (e.g., handles), agricultural products (e.g., reservoir liners), and novelties. Table III presents an end-use breakdown of the PVC resin market in 1973.

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TABLE II

PRODUCTION, PRICE, VALUE AND PRODUCERS OF POLYVINYL CHLORIDE RESIN

Production, Price, and Market Value

Year	Production (billion lbs)	Average Price (¢/lb)	Market Value ($ million)
1962	1.2	17.6	212
1967	2.1	15.6	327
1972	4.3	13.6	585
1973	4.5	14.5	650
1974	4.6	20.0	920

Producers and Capacity

Producer	Annual Capacity (million lbs) by December, 1975
Goodrich	950
Borden	545
Tenneco	480
Robintech	470
Continental Oil	430
Firestone Tire & Rubber	400
Diamond Shamrock	360
Union Carbide	350
Goodyear	300
Georgia Pacific	220
Others (Air Products, American Chemical, Certain-teed, Ethyl, General Tire, Olin, Pantasote, Shintech, Occidental Petroleum, Stauffer Chemical, Uniroyal, Keysor)	1,895
Total	6,400

Source: U.S. Tariff Commission and Arthur D. Little, Inc., estimates.

TABLE III

END USE BREAKDOWN OF PVC RESIN - 1973

End Use and Product	% of Market (Approximate)	Million Lbs (Approximate)
Building and Construction		
Pipe, conduit and fittings	29	1,345
Flooring	9	440
Wire and Cable Insulation[(a)]	8	365
Siding, panels	2	90
Wall covering	2	90
Other (window frames, trim, gasketing, sealants, miscellaneous)	2	90
Sub Totals	52	2,420
Home Furnishing and Household Goods		
Furniture (mainly upholstery)	7	320
Shower curtains, table covers, etc.	2	105
Appliance parts (including hoses, gaskets)	1	40
Garden hose	1	35
Other (e.g., window shades)	1	45
Sub Totals	12	545
Consumer Goods		
Phonograph Records	3	145
Footwear (shoes, rainwear)	3	140
Toys, dolls, and games	2	75
Outerwear (e.g., apparel)	2	70
Sporting and Athletic Goods (e.g., balls, bicycle grips, miscellaneous)	1	65
Other (including handbags, wallets, luggage)	1	55
Sub Totals	12	550
Transportation Equipment (mainly motor vehicles)		
Upholstery and Seat Covers	4	180
Wire and Cable Insulation	1	50
Auto Tops, headliners, trim, floor mats, other	1	45
Sub Totals	6	275

TABLE III (Cont'd.)

END USE BREAKDOWN OF PVC RESIN - 1973

End Use and Product	% of Market (Approximate)	Million Lbs
Packaging		
Flexible film (for fresh meats and produce)	3	135
Rigid Sheet (for cookies and candy, unit packs, blister packs)	2	105
Coatings (e.g., can linings, cap liners, gaskets)	1	50
Bottles, other	2	85
Sub Totals	8	375
All Other Uses		
Medical Products (e.g., tubing, blood bags)	1	45
Stationery Supplies	1	35
Agricultural Products (e.g., reservoir liners, tubing)	1	30
Novelties (including advertisement items)	1	30
Conveyor Belting	1	30
Miscellaneous (tools and hardware, credit cards, book binders and covers, other)	4	265
Sub Totals	9	435
Grand Totals	99	4,600 (b)

(a) Includes some wire and cable used in communications equipment.
(b) Domestic use only; excludes exports of about 145 million lbs.

Source: The Society of the Plastics Industry and Arthur D. Little, Inc., estimates.

As a result of this myriad of product and end-use applications, for the purposes of our impact analysis, we have segmented PVC resin usage, and grouped the end-use applications into five distinct modules. The modules and PVC end-use products included in each are given below.

(i) Building and Construction Module: pipe, conduit, floor covering, paint, insulated wire and cable, siding, panels, wall covering and other products (e.g., window frames, trim, gasketing);

(ii) Motor Vehicles Module: upholstery, insulated wire, seat covers, floor mats, dashboards, auto tops (e.g., landau types), and other "soft" component parts such as visors, head lining, and door panel covering, sealants and gaskets;

(iii) Home Furnishings, Household Goods and Apparel Module: upholstery for chairs and sofas, shower curtains, table covers, appliance parts, waterproof outerwear, window shades;

(iv) Specialty Products Module: phonograph records, telephone and telegraph equipment, and lighting and wiring equipment; and

(v) Miscellaneous Products Module: luggage, handbags, toys, sporting and athletic goods, footwear, garden hose, packaging materials, credit cards, medical products (tubing and other), stationery supplies, novelties, conveyor belting, tools and hardware and other.

Each of these modules is presented schematically in Appendix Figures A1 - A5.

There are literally thousands of business firms which fabricate PVC compound (which they make or buy as such) into semifinished and finished products. Such companies, which include nearly all of the PVC resin manufacturers, range in overall size from very large to very small businesses. Many others purchase such semifinished PVC based products and convert them (via cutting, sewing, heat sealing, etc.) into proprietary end products for sale as such to the final consumer. Table IV presents a representative list of major fabricators of PVC resin and compound and the main PVC-based products made by the companies.

TABLE IV

REPRESENTATIVE MAJOR FABRICATORS OF PVC

Company	Main PVC-Based Product(s) Made
Abbott Labs	Medical products (e.g., tubing, blood bags)
Andersen	Window frames
Armstrong Cork	Flooring
American Biltrite Rubber	Flooring, floor mats
Amerace-Esna	Hose, industrial parts
American Can	Can linings
Anaconda	Wire and cable
Avon Sole	Soles and heels
Borden[a][b]	Packaging film, upholstery, wall covering
Bata Shoe	Shoes, other footwear
Belden Manufacturing	Wire and cable
Bemis	Film and coated fabrics
Bird & Son	Siding
Congoleum Nairn (Bath Industries)	Flooring
Columbia Broadcasting System	Phonograph records
Capitol Records	Phonograph records
Certain-teed Products[a]	Pipe, siding
Chemical Products	Plastisols (custom formulated)
Chrysler	Auto upholstery, trim, sealants and gaskets
Clopay	Packaging film, window shades and wall partitions
Continental Can	Can linings
Crane Plastics	Trim, profiles
Dart Industries	Coated fabric, pipe, hose, industrial parts
Dayco	Hose, industrial parts, film
Diamond Shamrock[a]	Film and Coated Fabrics
Dennis Chemical	Plastisols
Essex International	Wire and cable
Evans Products	Trim, decorative laminates

TABLE IV (Cont'd.)

REPRESENTATIVE MAJOR FABRICATORS OF PVC

Company	Main PVC-Based Product(s) Made
Ethyl[a][b]	Pipe, bottles
Fields Plastics	Film and sheet
Firestone Tire & Rubber[a]	Film, coated fabrics, hose, industrial parts
Flintkote	Flooring
Ford Motor	Upholstery, trim, sealants
GAF	Flooring, siding
General Cable	Wire and cable
General Electric	Wire and cable
General Mills	Toys, dolls and games
General Motors	Wire and cable
General Tire[a]	Film, coated fabrics, hose, industrial parts, sporting and athletic goods
Genesco	Footwear
Genova	Hose, industrial products
Georgia Duck	Conveyor belting
Georgia-Pacific[a]	Pipe, decorative laminates
B. F. Goodrich[a][b]	Film, coated fabrics, hose, industrial parts, sporting and athletic goods, and plastisols
Goodyear Tire & Rubber[a]	Film, coated fabrics, hose, industrial parts, sporting and athletic goods, flooring, and packaging film
W. R. Grace	Film and coated fabrics, closure linings
Haartz-Mason	Coated fabrics (industrial)
Hooker Chemical (Div. Occidental Petroleum) (a)	Coated fabrics
Hoover	Hose and other appliance parts
Ideal Toy	Dolls, other toys
Indian Head	Pipe
Inmont	Coated fabrics
Johns-Manville	Flooring, siding, trim

TABLE IV (Cont'd.)

REPRESENTATIVE MAJOR FABRICATORS OF PVC

Company	Main PVC-Based Product(s) Made
Johnson & Johnson	Tapes, medical supplies (e.g., tape-based)
Johnson Plastic	Hose, industrial products
Kentile	Flooring
Keysor[a]	Phonograph records
Lilly, Eli	Bottles, other packaging products
Mattel	Toys (e.g., dolls)
Minnesota Mining	Tapes
New England Plastic	Profiles
Norton	Tubing (medical, other)
Olin[a]	Pipe
O'Sullivan Rubber	Footwear products (soles and heels), film, coated fabric
Owens-Illinois	Bottles, closure linings
Pantasote[a]	Film and coated fabrics, gasketing
Phelps-Dodge	Wire and cable
Plymouth Rubber	Film and coated fabrics, floor mat
Prevue Products	Footwear (e.g., waterproof)
R & G Sloane Manufacturing	Pipe and fittings
Radio Corp. of America	Phonograph records
Reichhold Chemicals	Custom compounds
Reynolds Metals	Packaging film
Robintech[a]	Pipe, siding
Rubbermaid	Household and automotive products
Rubatex	Hose, industrial parts
A. Schulman	Custom compound
Stauffer Chemical[a][b]	Upholstery, wall covering
Tenneco[a]	Film and sheet
Union Carbide[a]	Film and sheet
Uniroyal[a][b]	Upholstery, wall covering, film

TABLE IV (Cont'd.)

REPRESENTATIVE MAJOR FABRICATORS OF PVC

Company	Main PVC-Based Product(s) Made
Weymouth Art Leather	Coated Fabrics
Western Electric (American Tel. & Tel.)	Wire and cable

(a) Producer of PVC resin
(b) Producer of VCM

Source: Company and trade literature and Arthur D. Little, Inc.

C. Background on Input/Output Models

Input/Output is founded on the simple proposition that what goes into the production process must also come out and eventually finds its way to final consumers or demanders. Thus, one can define an industry's purchases (including materials, services, labor, capital and construction costs, as well as indirect costs such as taxes and profits) as equal to the sales of that industry. In other words, input is equal to output. The I/O accounting scheme is a systematic organization of each industry's purchases from, and sales to, every other industry, plus its sales to the domestic consumer, government, foreign consumer, and the investment and construction markets. Every sale by the selling industry is a purchase from the point of view of the purchasing industry. So each output from one industry can be viewed as a purchase by another. Business transactions among industries are organized in the analytical framework into a square "interindustry" or "flow" matrix, in which sales of each industry are shown along a horizontal row and purchases by each industry are found by reading down a column.

I/O analysis permits the systematic investigation of the inter-relationships of producing and consuming industries to final markets and primary inputs; thus, it is particularly useful for analyzing problems of those industries which primarily sell their output to other industries, and it permits the tracing of direct and indirect effects on total demand of changes in final goods markets.

For example, the Input/Output technique allows one to estimate the total impact upon various sectors of the economy of an increase in the demand for passenger cars. This increase in demand will lead to a direct increase in production of the automobile industry. However, there will be further impacts. The automobile industry will demand more upholstery fabrics and the increased production of these fabrics will require more synthetic fibers and more plastics. The use of Input/Output techniques allows one to quantify the magnitudes of the increased production in all affected industries.

In like fashion, the Input/Output methodology can be used to identify the impact of a reduction in the output levels of particular raw materials on the level of production in related final end-use markets. As in the case of PVC resin, the unavailability of this material will lead, for example, to production cutbacks in the automotive industry resulting from a lack of PVC-related products such as electrical wire, upholstery, and assorted component parts. However, cutbacks in automotive production permeate throughout the economy, generating lower demands for materials such as glass, steel, aluminum, rubber, etc. Lower demands in these industries would feedback into reduced outputs of iron ore, bauxite, petrochemicals, energy, etc. Thus, the total resulting economic impact would be far greater than the value of production and number of jobs lost in the automotive industry itself.

The circular flow and general equilibrium concepts of input/output analysis and its emphasis of interindustry relations can be traced back to Francois Quesnay's Tableau Economique. The first empirical application of the input/output model in the modern world dates from 1936 when Professor Wassily Leontief at Harvard University published an input/output system of the U.S. economy. Most recently, Dr. Leontief received the Nobel Prize in Economics, primarily for his work and contributions in the area of input/output analysis.

Even prior to the recognition of Leontief's work by the Nobel committee, a growing number of public and private organizations world-wide had already been using input/output tables and this conceptual technique in economic analysis, planning, and policy applications. In the U.S., the Department of Commerce has published benchmark input/output tables of the U.S. economy for 1947, 1954, 1958, 1963, and 1967; work is now underway on preparing a 1972 table. Recently, the Department of Commerce has announced that it will prepare input/output tables on an annual basis. This decision was based largely upon the growing use of input/output tables by other government agencies and also by private

corporations. In the public sector, agencies such as the Bureau of Economic Analysis, Bureau of Labor Statistics, Council of Economic Advisors, and Federal Energy Agency are using input/output tables and techniques in undertaking various analytical planning and policy efforts. The identification of input/output as a powerful tool in economic analysis and development planning and policy making has also greatly influenced many state and regional authorities. Today, more than 20 states and/or regions in the U.S. have constructed input/output models of their economies and work is currently on-going in several others. Internationally, the construction of input/output tables and their applications has increased rapidly. I/O tables now exist for most Western European nations and many African and Far Eastern countries.

In the private sector, domestic and foreign corporations have discovered the strengths of the input/output concept and with the availability of published Government tables and/or tables constructed internally by their staffs, corporations have applied input/output techniques in a whole host of efforts, including:

- economic impact analysis
- short- and long-term economic forecasting
- regional economic development and planning
- marketing and sales analysis
- diversification and acquisition studies

For the reader unfamiliar with input/output, there are several excellent texts which present a discussion of the concept, its mathematical foundation, and its possible areas of applications. Included among these are:

Almon, C., The American Economy to 1975. (New York: Harper and Row, 1966).

Chenery, H., and Clark, P., Interindustry Economics. (New York: John Wiley & Sons, Inc., 1959).

ONLY COPY AVAILABLE

Doofman, R., Samuelson, P., and Solow, R., Linear Programming and Economic Analysis. (New York: McGraw-Hill, 1958).

Miernyk, W., The Elements of Input-Output Analysis. (New York: Random House, 1965).

Richardson, H., Input-Output and Regional Economics. (New York: John Wiley & Sons, 1972).

Yan, C., Introduction to Input-Output Economics. (New York: Holt, Rinehart and Winston, 1969).

During its ten year association with input/output analysis, ADL has applied the technique in many of the above areas. In such efforts, we have combined the expertise of our in-house economists and industry specialists with that of recognized authorities on input/output to many universities. Indeed, Dr. Leontief has (and continues to) served as a consultant to ADL in many of our study efforts applying input/output techniques.

Much effort at ADL over the years has been expended in incorporating up-to-date interindustry relationships into the model in order that it more accurately identify the linkages which exist between sectors of the economy. Use of I/O tables that have not been kept current and thus do not reflect present market relationships would yield misleading estimates of the potential economic impact of a particular event.

A schematic representation of the complete ADL Input/Output model is presented in Figure 1. In the past several years, the model has been applied to several study efforts including:

- an assessment of the regional economic impact generated by the development of deep-water terminals and outer continental shelf drilling operations in the U.S.;

- an assessment of the economic impact resulting from a curtailment in petrochemical supply availability in the U.S.;

- an assessment of the economic impact of alternative federal government fiscal policy strategies; and

- an evaluation of the economic impact of alternative energy conservation programs.

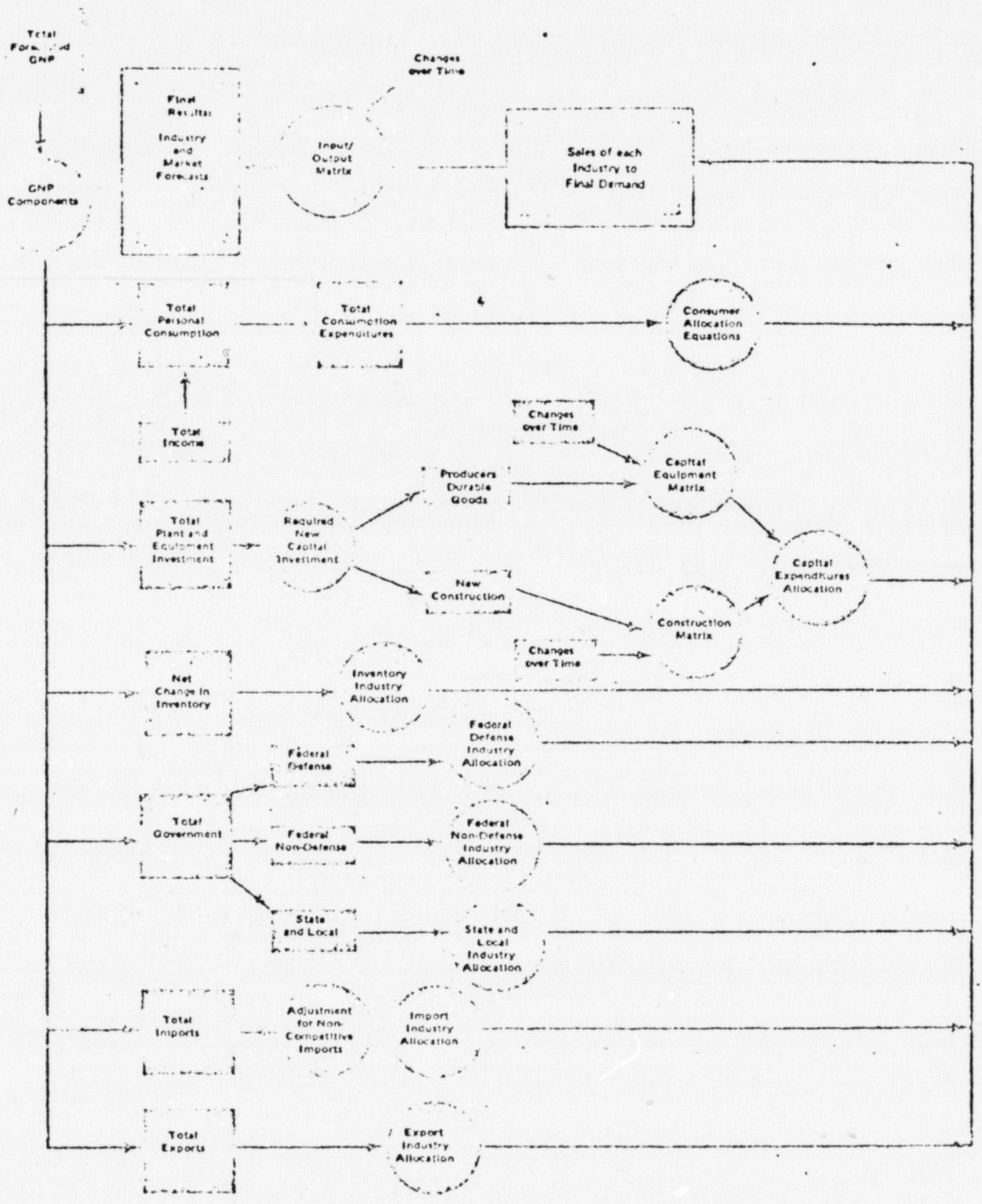

FIGURE 1 FLOW CHART OF ADL MODEL AND COMPONENTS

D. Study Results

The results of our basic impact analysis are summarized in Table V. Using the information on end-use applications of PVC resin collected as part of this effort, the ADL model was then run for each of the five modules to derive estimates of the resulting economic impact should PVC resin become unavailable.

In brief and in the jargon of input/output economics, the model is run given a new vector of final demands which reflect the unavailability of PVC resin in the end-use markets identified in each module. The matrix-vector multiplication which then follows yields estimates of the resulting impact upon all other sectors of the economy in terms of lost production and employment. Thus, the ADL model (and other current input/output models) can be used to trace through the effects of a lack of PVC resin in the automotive sector and estimate the impact not only upon auto-supplying industries such as steel, aluminum, rubber, glass, etc., but also upon those industries which supply necessary inputs for the production of these auto-related materials.

Tables A-1 through A-5 present a detailed breakdown of the economic impact for each module. Estimates are given for both the direct impact (e.g. automotive sector) and the indirect impact (e.g. all other sectors such as steel, glass, rubber, etc.). As the tables indicate, the magnitude of the economic impact is a function of:

- the size of the end-use industry;
- the extent of PVC dependency;
- the relationship between the end-use market and all other supplying industries. This relationship is an important product of an input-output table.

TABLE V

SUMMARY OF

ECONOMIC IMPACT OF IMMEDIATE

SHUTDOWN OF PVC RESIN OPERATIONS

Industry	Loss in Production (Billion $)	Loss in Employment (MM)
Vinyl Chloride Monomer	0.2	*
PVC Resin	0.6	**
Construction-Related	17.1-34.2	0.6-1.1
Home Furnishings, Household Goods and Apparel-Related	1.4--2.7	0.1
Motor Vehicle-Related	25.3-50.6	0.4-0.9
Specialty Products[1]	10.7-13.9	0.3-0.4
Miscellaneous Products[2]	3.6--5.7	0.1-0.2
TOTAL	$58.9-107.9	1.5-2.7
Inflationary Adjustment	+3.2-5.8	--
Calculated Range	$61-113	1.5-2.7
Most Probable Range[3]	$65-90	1.7-2.2

1. Includes products such as phonograph records, telephone and telegraph equipment, and lighting equipment.
2. Includes products such as luggage, toys, games, sporting goods, bottles, packaging film, credit cards, and medical tubing. Also includes chemical additives (e.g. plasticizer, stabilizers) used to make PVC-based compound and ethylene and chlorine used to make VCM.
3. The most probable range includes adjustments due to rounding, data estimating, and double counting.

* About 1,400 employees.
** About 5,000 employees.

Source: Arthur D. Little, Inc., estimates.

For example, in the automotive module, the input/output model estimates that for every dollar of production in the automotive sector, an additional $1.20 of production is required in all other sectors of the economy. It is not difficult to identify this $1.20 of other production as the demands placed upon producers of steel, aluminum, rubber, iron ore, coal, etc.

Every effort has been made in this analysis to present our best estimates of the potential impact and minimize any upward bias which could result from:

- double-counting - we have eliminated any double-counting which could result from two industry sectors supplying the same final market. For example, the automotive sector can be identified as an end-use market in both the automotive and apparel and home furnishings modules. In this case, the impact upon the sector was determined in the automotive module and not included in the apparel and home furnishings module calculation.

- overestimation of PVC dependency - our estimates of PVC dependency in each module is based upon our knowledge of the industry as defined in SIC terms and the extent of PVC resin usage in the particular industry. In several cases, because of a lack of information or because of the conventions adopted in the SIC codes, the extent of PVC dependency in a particular market was difficult to estimate. In all such cases, a "best estimate" was made with an intentional bias toward the conservative side. Note, for example, the relatively low level of PVC dependency assumed in the building and construction module.

- underestimation of PVC substitution possibilities - as the following chapter wi[illegible]al, much effort was expended in assessing the likely use o[illegible]stitute materials for PVC resins. In analyzing each PVC resin application, we assessed

the possibility of using substitutes realizing that present PVC resin users would strive to continue their operations by adopting alternative materials and processes.

As Table V indicates, the magnitude of the potential impact is quite significant, whether measured in terms of lost production or employment. The detailed data which underlies Table V is presented in Appendix A, Tables A-1 to A-5. The adjustment for inflation (Table V) has been included since the basic data used in our analysis has been taken from the 1972 Census of Manufactures. Therefore, in order to update the value of production data to reflect 1973 price levels, an upward adjustment was made based upon the average annual rate of increase in prices since 1972.

On balance, our analysis indicates that the resulting economic impact to the U.S. economy from an immediate shutdown of all PVC resin plants would equal approximately $65-90 billion of lost domestic production, and a reduction in employment of about 1.7-2.2 million. Because of the magnitude of economic activity and extent of PVC-related products usage in the building and construction and automotive sectors, these two segments of our economy would receive the greatest share of the total impact. Unavailability of PVC resin will have production bottleneck effects in these areas in terms of insulated wire and cable, flooring, upholstery, and many other materials.

E. Regional Impacts

In undertaking our analysis, we focused upon estimating the economic impact to the total U.S. nationally. However, as our effort proceeded, it became evident that the resultant economic impact could have a disproportionate effect upon several regions of the country which already have been suffering from depressed economic growth conditions and above-average unemployment rates. Recognizing this, we attempted to identify a small sample of those regions likely to be impacted severely by the shutdown of all PVC resin plants.

This brief analysis proceeded by estimating the percentage of a region's labor force presently employed in PVC-related industries and comparing this percentage with the current rate of unemployment in the area.* The number of employees in PVC-related industries was derived from data in County Business Patterns, 1972. However, in many cases, such information was withheld to avoid disclosure of certain operations and thus, the percentages derived in this brief analysis are in many cases conservative estimates. In addition, the percentages represent a minimum since we have not included employment in those industries indirectly linked to PVC-related markets. Nonetheless, based upon this limited analysis, several local areas were discovered as being likely to receive a severe economic impact. Table VI identifies several such areas and compares their present rates of unemployment with the percentage of PVC-related employment in their labor force.

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*PVC-related industries are those identified in each of the modules presented in Appendix Figures A1-A5.

TABLE VI

IMPACT ON SELECTED REGIONS

Area	April 1974 Unemployment Rate (%)	Department of Labor Designation *	Labor Force in PVC-Related Industries (%)
Massachusetts			
Worcester SMSA**	6.4	Substantial	5
New Bedford SMSA	8.9	Persistent	5
California			
Los Angeles SMSA	7.2	Substantial	6
Michigan			
Detroit	9.3	Substantial	20
Grand Rapids	9.1	Substantial	8

* Areas of Substantial Unemployment

A labor area in which the current and anticipated local labor supply substantially exceeds labor requirements is classified as an area of "substantial unemployment." An area is placed in this category when:

(1) Unemployment in the area is equal to 6 percent or more of its work force, discounting seasonal or temporary factors, and

(2) It is anticipated that the rate of unemployment during the next 2 months will remain at 6 percent or more, discounting temporary or seasonal factors.

Areas of Persistent Unemployment

A labor area, or a city of 250,000 or more population, or a county, may be classified as an area of "persistent unemployment" when unemployment during the most recent calendar year has averaged 6 percent or more of the work force, and the rate of unemployment has:

(1) Averaged 6 percent or more and has been at least 50 percent above the national average for 3 of the preceding 4 calendar years, or

(2) Averaged 6 percent or more and has been at least 75 percent above the national average for 2 of the preceding 3 calendar years, or

(3) Averaged 6 percent or more and has been at least 100 percent above the national average for 1 of the preceding 2 calendar years.

** SMSA - Standard Metropolitan Statistical Area.

Even this brief analysis does indicate that an immediate shutdown of all PVC resin plants in the U.S. would generate an economic hardship of large magnitude that would create severe economic dislocations in many local areas.

IV. PVC RESIN DEPENDENCY AND RAW MATERIAL SUBSTITUTION

A. PVC Resin Dependency

By PVC resin dependency we mean: to what extent is an industry or final product market sector dependent today on PVC resin as a raw material? For example, in the case of the phonograph records industry (SIC 3652), the PVC dependency is very high since nearly all phonograph records are based on PVC resin. On the other hand, in the case of the entire finished paint industry (SIC 2851), the PVC resin dependency is very low since the relative amount of PVC resin used is very small in comparison with the total amount of synthetic resin used by that industry.

The question of dependency depends upon the definition of the particular industry sector being considered. For example, the relative PVC resin dependency of all pipe production is quite small since most pipe is made out of metal, concrete and other non-plastic materials. However, if the industry being evaluated is the plastic pipe industry, then the PVC dependency is high since PVC represents about 70% of the total weight of resin used to make plastic pipe.

PVC resin dependency varies widely in the industries and market sectors that are contained in our five modules. In Table VII we have listed the PVC resin dependency for the major industries and end uses of significance to this study. If PVC resin is the major raw material used in a given industry or product sector, the dependency rating (as expressed as an approximate percent of the total resin based raw materials used) is high. Conversely it is given a low dependency rating if PVC is not a primary raw material in that particular industry. It should be noted, however, that even though the PVC resin dependency may be low, as in the motor vehicles industry (since PVC resin represents only about 0.5% of the weight of a typical automobile), if products made from PVC are absolutely necessary in the manufacturing process of that industry, the unavailability of those PVC products could result in a major shutdown of that industry. This would be the case with the original equipment manufacture of motor vehicles, with the

TABLE VII

PVC RESIN DEPENDENCY IN SELECTED INDUSTRIES

Module and Industry	SIC Number	PVC Resin Dependency (%)
Building and Construction		
All pipe, conduit and fittings	--	small
Plastic pipe, conduit and fittings	Part of 3079	70
Flooring (hard surface only)	Part of 3079	90
Wire and cable insulation	Part of 3357	40
All siding	--	small
Wall covering	2644	40
Paint	2851	small
Motor Vehicles		
Upholstery material, trim, external tops, head liner, door covering	--	90
Wire and cable insulation	Part of 3357	50
Sealants and gaskets	--	25
Household Products and Apparel		
Furniture upholstery	--	10
Table covers, shower curtains, place mats	--	75
Garden hose	Part of 3079	50
Outerwear, other apparel	--	small
Raincoats, other waterproof outerwear	2385	10
Curtains and draperies	2391	5
Household tapes	--	50
Baby pants	--	90
Carpet backing (foamed underlay)	--	25
Window shades	--	90
Specialty Products		
Phonograph records	3652	90
Communication wire and cable	Part of 3661	75
Electric lighting & wiring equipment	364	small
Metal cans (e.g., for all beverages)	3411	small
Glass containers (e.g., for baby foods)	3221	small
Conveyor belting	--	25

TABLE VII (Continued)

Module and Industry	SIC Number	PVC Resin Dependency
Miscellaneous Products		
Luggage	3161	50
Handbags and purses	3171	75
Toys and dolls and games	3941, 3942	10
Plastic bottles	Part of 3079	10
Flexible packaging material (film)	Part of 3079	10
Credit cards	--	75
Medical tubing, blood bags	--	90

Source: Arthur D. Little, Inc., estimates.

construction of houses and other buildings (e.g., caused by a lack of PVC insulated building and communication wire and flooring materials) and in the packaging and storage of beer and soft drinks in cans (all have PVC-based linings) and other food products, such as baby foods, in glass jars (all have vinyl gaskets inside the covers).

B. Substitution of Other Raw Materials for PVC Resin

Essentially all major classes of products now made from PVC resin were made on a commercial scale from other raw materials prior to the introduction of PVC resin in the U.S. in 1939. It would seem, therefore, that it should be possible to use substitute raw materials in place of PVC resin. However, because of the unique nature of PVC resin which, when combined with many other raw materials, permits the formulation of hundreds of different compounds with wide ranges of properties (from very soft to very hard materials), and colors, it would not be possible, in fact, to duplicate the exact properties of vinyl plastic products by using substitute raw materials. Even if it were possible from a physical property and decorative point of view to make an "equivalent or acceptable" product derived from non-PVC resin-based materials, the costs of production and the resulting selling prices of the final products would in essentially all instances be greater than those products made from PVC. Such costs and prices would probably be prohibitive in many cases and in effect price the substitute final product "out of the market."

Assuming, however, that (a) the substitute raw materials--some of which are now made in small quantities relative to PVC and in very short supply (including a very low level of inventory) and are likely to remain in "tight" supply for at least 2-3 years (such as other plastics, rubbers, metals, paper, textile products, and glass)--will be available in sufficient quantity when needed, and (b) the physical capacity to produce the additional non-vinyl chloride based end products is also in-place and operating when required (which is most unlikely), it would still take up to an additional three years to complete all of the necessary research and development leading to the required changes in product, process, and equipment design and the actual delivery and installation of new equipment in order to make such non-vinyl equivalent products on a commercial scale necessary to satisfy the demands

of industry. This latter stage of the manufacturing process (after the product and process design changes have been made) could in itself take up to two years for many of these "new" products. Based solely on technological factors, those substitute products which would take the longest lead time to develop are those that are critical products particularly for the construction industry and motor vehicle industries. Such products include: insulated communication, building and automotive wire and cable, pipe, flooring, and automotive upholstery and related soft trim materials.

A discussion of the PVC substitution issues according to several of the major PVC product lines is presented below.

1. Pipe, Conduit and Fittings

This is by far the largest single PVC market accounting for about 29% of current total PVC resin consumption in this country. Over two-thirds of all plastic pipe and fittings is based on PVC with ABS and polyethylene plastic each representing about 15%. Most of the other 5% is accounted for by rubber-modified styrene type resins. Roughly 65% of all PVC pipe is used in pressure applications such as for use in chemical processing plants. In these applications PVC is used for its chemical and corrosion resistance, its non-flammable properties, its rigidity and ease of installation as well as its relatively low price. Polyethylene is also used in some of these pressure pipe markets (e.g., gas distribution) but does not compete directly with PVC in most applications. ABS resin is not used in these particular pipe markets mainly because of its higher price and lack of chemical and flame resistance. ABS, however, is a major competitive plastic for PVC in DWV (drain, waste and vent) pipe for home construction and also competes with PVC in electrical and communication conduit and sewer pipe. It is possible that ABS plastic, in particular, could be substituted in 20-25% of the PVC pipe market; however, this would increase the total ABS resin market by roughly one-third and the resin would not be available in this quantity for this market for at least 2-3 years. Conceivably some of the PVC pipe

markets could be satisfied from a property point of view by metal pipe. However, the metal pipe industry is itself operating at capacity and likewise could not supply the replaced PVC pipe market in less than two years.

2. Upholstery Material (coated fabrics)

(a) Automotive

Most motor vehicles today use vinyl-coated fabrics as the primary upholstery material; the backing material (fabric) is still largely cotton, but in the future, synthetic fiber based materials will be used to a greater extent. The vinyl coating is applied either by calendering or plastisol casting operations. Calendering is by far the major method of fabrication used for these products; only about 15% of automotive upholstery coated fabrics are made by the casting (or knife coating) process. The casting process is generally more expensive than calendering because (1) it uses more costly PVC (plastisol) compounds, and (2) production-line speeds are much lower than for calendering lines--30-50 yds/min. vs. 70-100 yds/min. The casting process is used primarily for low-volume, short-run operations.

In addition to PVC-coated fabrics, some motor vehicles are still made using conventional uncoated textile upholstery fabrics such as those based on nylon and polyester blends; however, today, these all-textile fabrics represent only a small percentage of the market. Even assuming that the nylon and polyester fibers were available in ample supply, the textile products industry would still need at least two years to install the necessary equipment to manufacture such quantities of upholstery fabrics from synthetic fibers as required by the auto producers.

In the absence of PVC, the preferred approach would be to substitute another synthetic resin such as polyurethane. However, polyurethane-based coated fabrics are not easily calendered as are the PVC based products. Coated polyurethane fabrics made by the casting process are now used in the apparel and furniture upholstery markets, where they are considered "deluxe" products. Also, polyurethane coated fabrics are now being used selectively in the automotive industry in Europe, and they are presently being tested by the U.S. auto makers.

According to our industry contacts, polyurethane-coated fabrics should have adequate properties to essentially meet the performance required of them in this application. Polyurethanes, in general, lack good UV stability; but now some materials are available that can meet this requirement. In some property aspects, polyurethanes are superior to PVC. For example, they have better abrasion resistance; consequently, thinner coatings can be used to produce equivalent properties when compared to PVC.

If polyurethanes are coated onto fabrics by the casting process, a research and design period of about one year would be required to meet the needs of the motor vehicle industry. However, casting is not the process used by most of the coated fabrics industry and several new facilities using different equipment would have to be constructed. For example, the urethane casting process requires significant drying facilities such as ovens, because the polyurethanes are applied as a solution from which the solvent must be removed (this is not the case with PVC plastisol casting). Disregarding polyurethane material shortages which are significant, 18-24 months would be needed to install the necessary casting facilities to "handle" this market. Moreover, because the production rate for the casting process is considerably slower than the calendering process, relatively more casting facilities would be needed in place of the existing PVC calendering facilities to meet demand. Thus, from a technology viewpoint only, the casting-process approach could be a reality in about 2-3 years.

On the other hand, if the polyurethanes were processed by the calendering process, then the research and design period itself would last as much as two years. While a few polyurethanes are available today that can be calendered, more research will be needed to develop the specific polyurethanes needed by the motor-vehicle industry. Therefore, if the calendering process were selected as the preferred approach, then the polyurethane substitute coated fabric could be at least four years away from commercialization. This approach might be preferred because, as mentioned above, most present fabricators of PVC-coated fabrics use calendering equipment. However, most industry respondents agree that polyurethanes designed for calendering would process at a slower production rate than PVC. Also,

because the price of urethane compounds is about four times that of PVC compound, the price of urethane-based coated fabric would be substantially higher than PVC-coated fabrics.

Chlorinated polyethylene (CPE) is another possible substitute material, although at present its absolute output and availability is very limited. This product has the advantage that it can be calendered in much the same manner as the present calender-grade PVC, and some fabricators have already worked with this resin. In their opinion, the product is close to meeting existing PVC specifications for automotive upholstery. However, CPE does not have the necessary low-temperature flexibility needed by the motor vehicle manufacturers. Furthermore, it is more difficult to calender than the PVC compounds; some have estimated that the production rate would be slowed by as much as 20% compared to PVC. Still another possible substitute for PVC in this application is ethylene-vinyl acetate copolymer (EVA). This product also can be calendered but, again, it lacks the necessary low-temperature flexibility. Because EVA, in contrast to PVC and CPE, lacks inherent flame resistance, it has to be especially formulated to meet this requirement. Thus, substituting EVA or CPE would mean some sacrificing of performance. Furthermore, these substitute products would still require about three years to reach commercialization; again, assuming the raw materials were available.

Fabricators and motor vehicle manufacturers are also developing new methods for manufacturing motor vehicle seats--methods that would not require coated fabrics. For example, one promising approach is the manufacture of one-piece molded seats using foamed polyurethanes with an external skin; the boating industry is currently using products of this type. However, this approach will require considerably more research and development time than the approaches described above in terms of replacing PVC-based automotive upholstery fabrics.

(b) Furniture

Today, polyurethane coated fabrics are used commercially as furniture upholstery material. However, it is still a small part of the market compared

to PVC-coated fabrics. At the present time, most of these coated fabrics are made by the solution casting process. As mentioned above, a series of relatively new urethane polymers have become available that can be calendered. In either case, the preferred substitute for PVC in this application is the polyurethane family of polymers, whether the manufacturer's existing facilities include casting or calendering equipment. The product design period for the changeover in this market should be no more than one year, and following that the modification of the existing equipment and the installation of some new equipment also should take place within one additional year. Thus, the time lag for PVC substitution in this application would be about two years, assuming that the polyurethane materials were then available. Manufacturers of PVC based furniture upholstery, like automotive upholstery, have also considered other substitute materials. For example, ethylene-vinyl acetate polymer is another candidate material. Though it can be calendered and formulated to meet most property requirements, it lacks low-temperature flexibility. Chlorinated polyethylene has been used, but it would have similar deficiencies.

3. Flooring

Nearly all of the hard surface (resilient) flooring products made today are based on PVC resin. The production of asphalt tile (made in only a limited color range) has been largely supplanted by vinyl-asbestos type and the output of linoleum and felt-back enameled "rugs" has decreased considerably relative to the past. In fact, many of the plants that used to make these latter products have been converted to PVC-based flooring products or dismantled entirely.

Because of the unusual ability of PVC resin to be made in a variety of colors (including pastels), surface finishes ("shiny" or dull), degrees of hardness, and the fact that PVC possesses excellent chemical and flame resistance, it would essentially be impossible to duplicate the same line of vinyl flooring products now on the market by using other synthetic or natural polymers. New facilities would have to be built to produce much larger quantities of linoleum and asphalt tile if the consumer would indeed "go back" to using these inferior products. It is more likely that most of the hard surface plastic flooring market would

be replaced by soft carpeting rather than with these "outmoded" resilient flooring materials.

4. Wire and Cable

(a) Building Wire

This wire carries a maximum voltage of 600 volts; typically, manufacturers sell 110, 220, and 440-voltage wire to industrial, residential, and commercial users. In this market, a PVC compound is most often used as the insulation material.

PVC was originally selected as the preferred material for this application because of its low cost (which derives in part from its "easy" processability), excellent flame resistance, good low-temperature flexibility and colorability (e.g., for coding) in addition to desirable electrical insulation properties. If PVC were no longer available, a major constraint that would inhibit the introduction of a substitute material would be the existing building codes. Although most of the codes involve performance specifications, they essentially restrict the material to PVC, because they specify performance requirements that only PVC can meet, such as flame resistance and flexibility.

The "easiest" substitute approach would be to use polyethylene plastics. Although this material lacks the inherent flame resistance of PVC, the industry believes that given sufficient time it could be formulated to meet this requirement. However, polyethylene does not satisfy the operating temperatures required in many building-wire applications. Although "flame-resistant" polyethylene could be used in most homes, it would probably be unsatisfactory in many industrial, commercial, and institutional applications where relatively high service temperatures are required. Polyethylene is also less flexible than PVC, a major disadvantage in household wiring; one approach might be to use thinner coatings to get improved flexibility; however, this might result in unsatisfactory insulation value.

Another PVC substitution approach would be to use EP (ethylene-propylene) rubber (EPR) with an outer coating of HYTALON (chlorosulfonated polyethylene)

or neoprene. By itself, EPR is too soft for many applications and does not have the required flame resistance; therefore, the HYPALON or neoprene coating would be required. These materials would impart the necessary flame resistance. Another approach would be to use HYPALON or neoprene only, disregarding the much higher cost and the lack of availability. Although neoprene would be satisfactory in many building wire applications, it lacks the necessary abrasion resistance required in several non-building construction applications. Another potential substitute material is crosslinked polyethylene (XPE), a thermosetting material. Again, it would have to be formulated to meet the flame resistance requirements; otherwise, it would have all of the other necessary properties, including the relatively high-service-temperature property. XPE, however, is a rigid material which would limit its use significantly in this market.

Assuming that the constraint of the building codes was removed and that sufficient amounts of the substitute materials were available (again essentially impossible from a physical capacity viewpoint), the redesigning of the substitute product would take up to two years, depending upon which substitute materials were selected. But even if the "easiest" approach were taken, e.g., using polyethylene, the existing extruding equipment would have to be modified (e.g., new and different screws would be needed). If a thermoset material, such as HYPALON, were selected, then considerable new auxiliary equipment and facilities also would be required. Therefore, depending upon which material was selected, the total time needed to commercialize substitute building wire products would be from two to four years.

(b) Automotive Wire and Cable

The average passenger car uses about ten pounds of PVC compound as insulating material for wire and cable; although small in terms of weight percent, this is a very necessary product that is used in motor vehicle production. According to the industry, the best substitute material would be polyethylene. Ordinary polyethylene has an operating temperature somewhat lower than PVC; therefore, in most instances, the auto industry would rather use crosslinked polyethylene which is used in some automotive wire applications today. Whereas PVC has a rated operating temperature of 105°C, the

crosslinked variety of polyethylene has an operating temperature of 150°C. Although polyethylene itself will not meet the existing flame-resistance requirements, polyethylene can be satisfactorily formulated to meet these requirements. However, substituting polyethylene would mean giving up other performance requirements such as flexibility.

Because crosslinked polyethylene wire is a commercial product today, the redesign period would be relatively short--perhaps about six months--if crosslinked polyethylene were used as a substitute. However, this material is processed differently than is PVC, and the need for additional equipment and space would introduce an additional time lag of 12-24 months. While the existing PVC extruders could be used in the fabricating operation, the processors would have to change the extruder screw but, more significantly, they would have to add new curing lines. Curing of crosslinking polyethylene requires heat and, therefore, more space and obviously more steam would be needed than is the case with PVC products. Furthermore, substituting crosslinked polyethylene would also reduce productivity--perhaps by as much as one-third. Consequently, additional extruding equipment also would be needed. In summary, substituting XPE for PVC in automotive wire applications would require about two years.

(c) Communication Wire

Flexible PVC compound is widely used as the wire insulation and jacket material for communication wire. This insulation is primarily used inside buildings--commercial, industrial, and residential. As a consequence, this insulation material must meet local building codes. PVC is the preferred material in this application because of its overall cost/performance characteristics, its relatively high-temperature resistance, and its good flexibility and colorability. Perhaps most importantly because it is used inside buildings, it is preferred because it meets the necessary flame-resistance requirements. In some applications where PVC is used currently, the industry could substitute polyethylene, if it were formulated to meet the flame-resistance requirement. On the other hand, neoprene could probably meet all of the existing requirements met by PVC insulation today, even though neoprene's electrical properties are slightly inferior to those of PVC.

HYPALON also can be considered a good substitute material. But HYPALON and neoprene rubbers are thermoset materials that would require new processing equipment. A thermoplastic material would be preferred. For example, chlorinated polyethylene might be used, if it were available in the quantities needed to satisfy this large market. The best approach would be to substitute appropriately formulated polyethylene for many of the current applications that use PVC and use neoprene as the substitute material for those applications that cannot be served by polyethylene alone.

Again, disregarding the constraint introduced by the need to change the existing building code requirements, extensive research and development would be needed to develop the substitute insulation material for communication wire. The specifications for materials used in this application are very stringent. Thus, R&D required to develop a neoprene substitute product would probably involve at least a two-year period. Moreover, as mentioned above, neoprene would require different

processing equipment. Therefore, another two years would be needed to install the new facilities and build the additional space to manufacture the neoprene-insulated wire. The design and development period for the polyethylene substitute material would be somewhat shorter. Consequently, the timing for substituting PVC in this application would be from three to four years.

5. Packaging

(a) Meat Wrapping

At the present time, flexible PVC is essentially the only wrapping film used for fresh meat. In the past, cellophane was used; consequently, cellophane is a potential substitute material. However, the manufacturing process required to make cellophane film is completely different from that used to fabricate PVC film. New facilities might be needed to manufacture the additional cellophane film, because at the present time most cellophane manufacturing facilities are relatively antiquated. Two to three years would be needed to construct these facilities.

Cellophane would meet most of the existing performance requirements; however, it is not as flexible as PVC. Other substitutes for PVC in this application might be polyethylene and ethylene-vinyl acetate resins. However, these alternatives are not very acceptable, because they are inferior to PVC with respect to clarity and oxygen transmission properties.

(b) Can Coatings

Most metal cans have an internal coating to protect the contents of the can from metal contamination. In some instances, these coatings also protect the metal from corrosion. In food canning applications, a variety of coatings are used, including vinyl chloride based copolymers. Other coating materials used for these purposes include oleo resins, phenolics, and epoxy resins; also some polybutadiene resins are used in beverage cans.

Polyvinyl chloride based resins have been used primarily because of their flexibility--a property that is important in the manufacture of two-piece metal cans that are "deep drawn." The industry could use epoxy resins as a substitute product in this application, because it would require a minimum of new equipment. The epoxy coating could be used in beverage and food cans. However, the use of epoxy resins, will slow production somewhat for it is more difficult to spray; most two-piece cans are sprayed while three-piece cans are coated by a roll coating process. The epoxy resins also may introduce a flavor problem. From this point of view, polybutadiene coatings would likely be preferred for beverage containers because they impart little or no taste. Assuming that epoxy resins were commercially available, the time required for the changeover would be minimal--less than one year.

PVC coatings are also used in the manufacture of some composite (paper-foil) cans used for foods. Here, it is typically used as a slip coating on aluminum foil. It also provides heat sealability. In this case, the industry has substitute products under development and, if PVC were not available, these products could be introduced within 6-12 months.

(c) Crowns and Closures

Most beverage crowns use a PVC plastisol compound as an insert to provide the necessary seal to keep the content of the bottle fresh for long periods of time. The use of cork, the original product, for this application represents no more than 10% of this market now; cork, which is imported, is very difficult to obtain and is expensive. The new convenience "roll-on" closures for beverage bottles also use a plastisol compound as the liner material. Although cork is a substitute material for this application, realistically, it is not preferred. The use of a cork liner requires very different equipment than that which is

used to apply the plastisol liners. At the present time, substitute products are under development, and one manufacturer has developed a new ethylene-based elastomer material. This product is currently in use in Europe. However, to utilize this new material, new equipment would be required and installation of this equipment would introduce a time lag of about one year.

Plastisol materials are also widely used as liners for the wide-mouth jar closures. For example, most vacuum-packed food jars use plastisol liners. In this case, the best substitute product would be a rubber latex material. Usually a natural or an SBR rubber is used. However, for the most part, these products would not meet the existing performance requirements. Although rubber does have adequate sealing capabilities in many applications, there are some applications where it does not. Also, in some of the high temperature processes required during the bottling of food, rubber is not as good as plastisol. In addition, rubber often has "cut through" problems and can introduce taste problems.

However, some rubber latices are presently used in this application, and therefore, a changeover to this substitute material could be carried out with a minimum of research and development time. (Sealing products in these applications, of course, must meet FDA requirements.) The equipment required to manufacture closure liners based on rubber latex is different than that required for the manufacture of plastisol liners. Consequently, about one to two years would be required to obtain the necessary equipment in-place. Furthermore, the process that uses rubber latex is considerably slower than that used for plastisol. Therefore, the production rate would be cut substantially.

In the case of non-vacuum closures, substitution will be much easier. Here, plastisol is not used as widely, and rubber or coated paper inserts can provide satisfactory performance. Table VIII presents a summary of the primary substitution materials for PVC resin and the time required to substitute them in the different major end uses.

TABLE VIII

SUBSTITUTION ASPECTS OF FABRICATED VINYL PRODUCTS

PVC Based Product	Primary Substitute Raw Material Candidates	Total Time Required for Substitution[a] (years)
Pipe	ABS, Polyethylene Metal	1-2
Conduit	ABS, Polyethylene Metal	1-2
Flooring		
Tile	Coumarone-indene resin, SBR	2-3
Yard Goods	Linseed oil (for linoleum)	3-4
Upholstery Material		
(Coated fabrics)	Polyurethane, CPE	
Automotive		2-3
Furniture		1-2
Wire Insulation	Polyethylene, Neoprene, HYPALON, EP Rubber	2-4
Phonograph Records	Polystyrene	3-4
Siding	Wood, metal (steel, aluminum)	1-2
Packaging Materials		
Flexible Film	Cellophane	1-2
Rigid Film	Cellulosic resin, polystyrene, nitrile resin	1-2
Bottles	Nitrile resin, glass	1-2
Cap liners	Cork, rubber	1-2
Can linings	Epoxy resin	1-2
Medical Tubing	Rubber (e.g., thermoplastic elastomer)	1-2

(a) Assumes that (1) sufficient quantities of substitute raw materials would be available when required by market demand; (2) production facilities are in place and operating at time required.

Source: Arthur D. Little, Inc., estimates.

FIGURE A-1

BUILDING/CONSTRUCTION MODULE

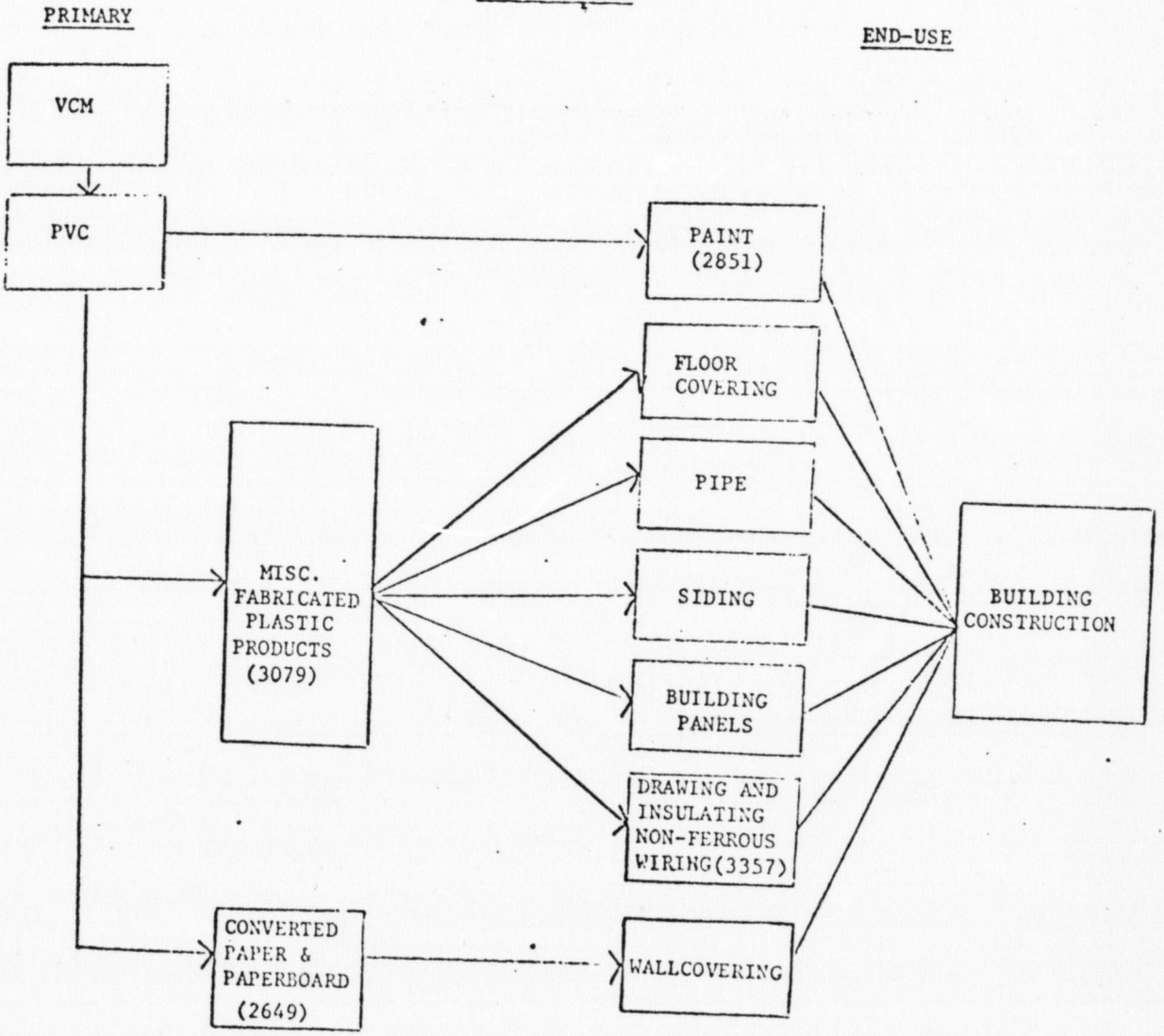

FIGURE A-2

HOME FURNISHINGS AND HOUSEHOLD GOODS AND APPAREL MODULE

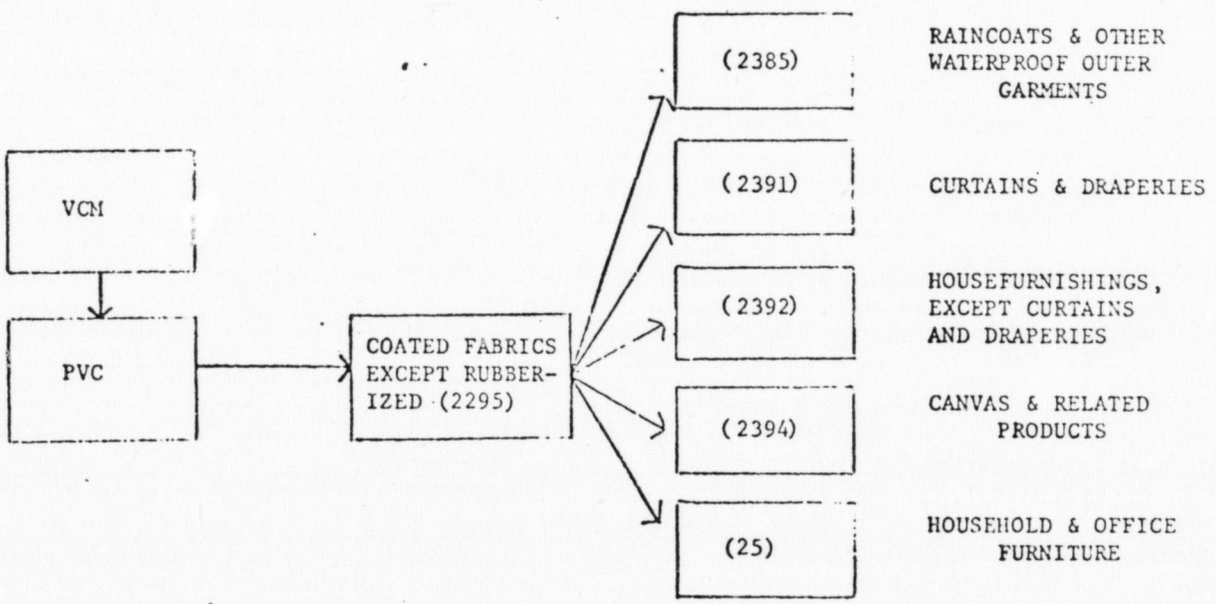

FIGURE A-3

MOTOR VEHICLES MODULE

INTERMEDIATE

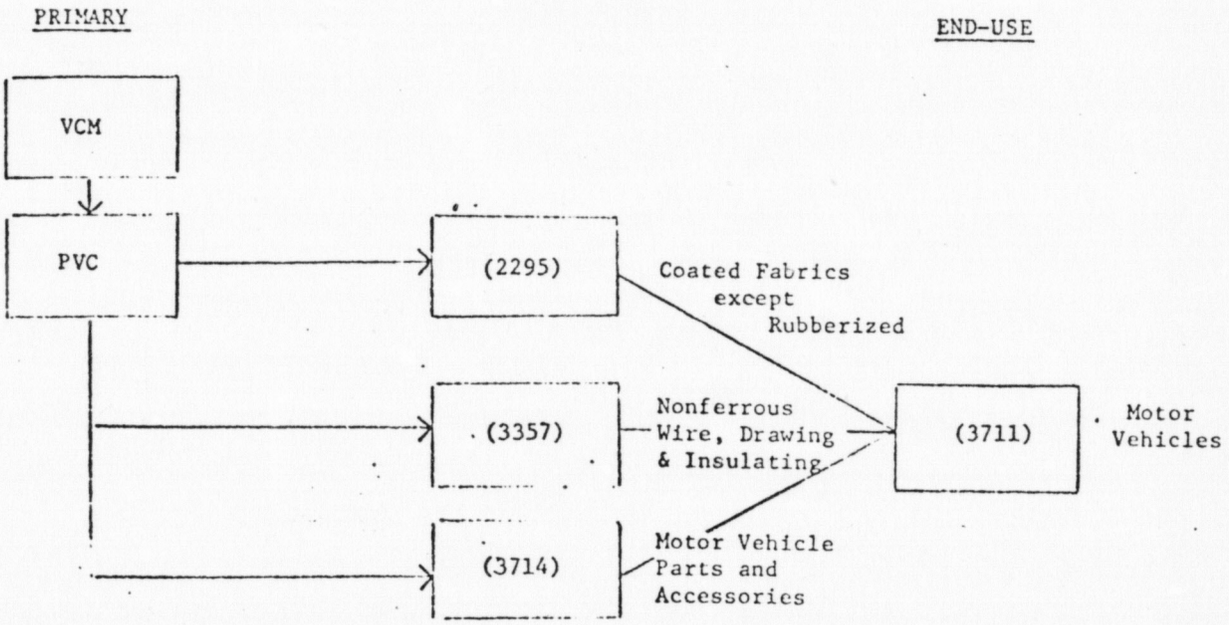

FIGURE A-4

SPECIALTY PRODUCTS MODULE

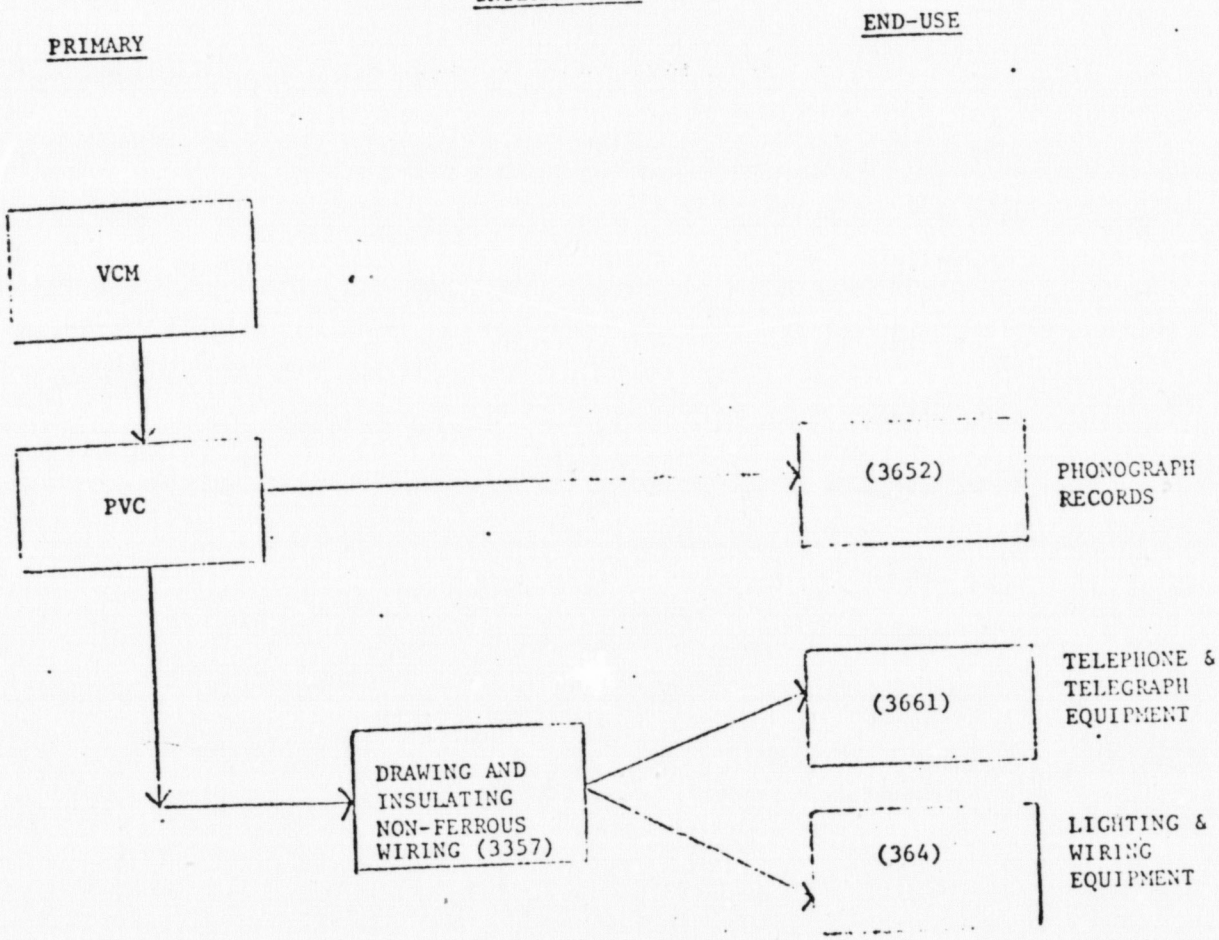

FIGURE A-5

MISCELLANEOUS PRODUCTS MODULE

INTERMEDIATE

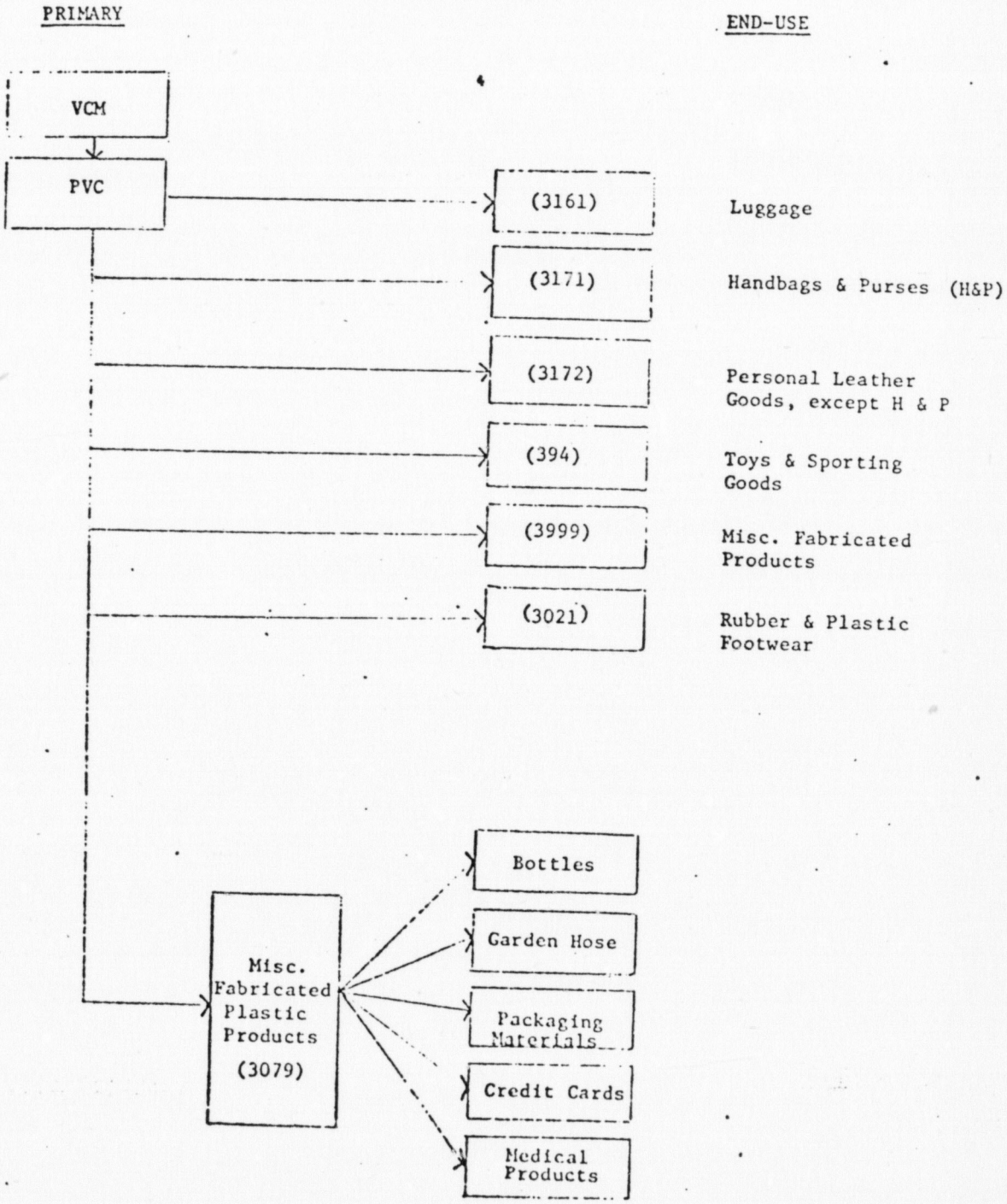

TABLE A-1

Building/Construction Module

Industry	1972 Value of Shipments (MM$)	1972 Employment (000)	Estimated PVC Dependency	Direct Loss in Shipments (MM$)	Direct Loss in Employment (000)	Impact on All Other Industries Loss in Shipments (MM$)	Impact on All Other Industries Loss in Employment (000)
Primary							
VCM							
PVC							
Intermediate							
Paint (2851)	3856	68					
Misc. Fabricated Plastic Products (3079)	9155	303					
Floor Covering							
Pipe, Conduit & Fittings							
Siding							
Building Panels							
Drawing and Insulating Non-Ferrous Wiring (3357)	4177	65					
Wall Covering	79	3					
End-Use							
Building/Construction	95000	3521	10%-20%*	9500-19000	352-704	7600-15200	190-380
TOTAL				$9500-19000	352-704	$7600-15200	190-380

TOTAL Modular Impact	Production (Billion $)	Employment (Million)
Direct	$9.5-19.0	0.4-0.7
All Other	7.6-15.2	0.2-0.4
	$17.1-34.2	0.6-1.1

* This estimate of PVC dependency represents a "best conservative" estimate based upon our work effort. The dependency could be greater given the high level of importance of such PVC-related items as building wire, pipe, and floor covering in many segments of the building/construction industry.

Source: 1972 Census of Manufactures and ADL estimates.

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TABLE A-2

Home Furnishings, Household Goods and Apparel Module

Industry	1972 Value of Shipments (MM$)	1972 Employment (000)	Estimated PVC Dependency	Direct Loss in Shipments (MM$)	Direct Loss in Employment (000)	Impact on All Other Industries Loss in Shipments (MM$)	Impact on All Other Industries Loss in Employment (000)
Primary							
VCM							
PVC							
Intermediate							
Coated Fabrics except Rubberized (2295)[a]	792	17					
End-Use							
Raincoats & Other Waterproof Outer Garments (2385)	288	14	5%-10%	14-28	0.1	13-25	*
Curtains & Draperies (2391)	745	33	5%-10%	37-74	0.1-0.3	33-66	*
Housefurnishings (2392)	1372	48	5%-10%	68-137	0.2-0.5	61-123	*
Canvas & Related Products (2394)	245	11	5%-10%	12-24	0.1	11-22	*
Furniture (25)	11231	460	5%-10%	561-1123	23.0-46.0	533-1066	13-27
TOTAL				692-1386	23.5-47.0	651-1302	13-27

(a) e.g., furniture upholstery material.

TOTAL Modular Impact	Production (Billion $)	Employment (Million)
Direct	0.7-1.4	0.1
All Other	0.7-1.3	*
	1.4-2.7	0.6-1.1

* Less than 50,000.

Source: 1972 Census of Manufactures and ADL estimates.

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TABLE A-3

Motor Vehicle Module

Industry	1972 Value of Shipments (MM$)	1972 Employment (000)	Estimated PVC Dependency	Direct Loss in Shipments (MM$)	Direct Loss in Employment (000)	Impact on All Other Industries Loss in Shipments (MM$)	Impact on All Other Industries Loss in Employment (000)
Primary							
VCM							
PVC							
Intermediate							
Coated Fabrics (2295) (a)	792	17					
Nonferrous Wire Drawing and Insulating (3357)	4177	65					
Motor Vehicle Parts and Accessories (3714)	18554	401					
End-Use							
Motor Vehicles (3711)	45979	359	25%-50%	11494-22988	90-180	13793-27585	345-689
TOTAL				11494-22988	90-180	13793-27585	345-689

TOTAL Modular Impact	Production (Billion $)	Employment (Million)
Direct	11.5-23.0	0.1-0.2
All Other	13.8-27.6	0.3-0.7
	25.3-50.6	0.4-0.9

(a) e.g., upholstery materials.

Source: 1972 Census of Manufactures and ADL estimates.

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TABLE A-4

Specialty Products Module

Industry	1972 Value of Shipments (MM$)	1972 Employment (000)	Estimated PVC Dependency	Direct Loss in Shipments (MM$)	Direct Loss in Employment (000)	Impact on All Other Industries Loss in Shipments (MM$)	Impact on All Other Industries Loss in Employment (000)
Primary							
VCM							
PVC							
Intermediate							
Drawing and Insulating Non-Ferrous Wiring (3357)	4177	65					
End-Use							
Phonograph Records (3652)	472	18	80%-90%	378-425	14-16	340-382	8-9
Telephone & Telegraph Equipment (3661)	4301	138	65%-75%	2796-3228	90-104	2237-2582	56-64
Lighting & Wiring Equipment (364)	5466	168	50%-75%	2733-4100	84-126	2186-3280	55-82
TOTAL				5907-7753	188-246	4763-6244	119-155

TOTAL Modular Impact	Production (Billion $)	Employment (Million)
Direct	5.9-7.7	0.2-0.2
All Other	4.8-6.2	0.1-0.2
	10.7-13.9	0.3-0.4

Source: 1972 Census of Manufactures and ADL estimates.

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TABLE A-5

Miscellaneous Products Module

Industry	1972 Value of Shipments (MM$)	1972 Employment (000)	Estimated PVC Dependency	Direct Loss in Shipments (MM$)	Direct Loss in Employment (000)	Impact on All Other Industries Loss in Shipments (MM$)	Impact on All Other Industries Loss in Employment (000)
Primary							
VCM							
PVC							
End-Use							
Luggage (3161)	345	16	50%-75%	172-258	8-12	206-310	5-8
Handbags & Purses (3171)	313	21	50%-75%	156-234	11-15	187-280	5-7
Personal Leather Goods, except H&P (3172)	274	13	50%-75%	137-205	6-10	164-246	4-6
Toys & Sporting Goods (394)	2800	100	30%-40%	840-1120	30-40	756-1008	19-25
Misc. Fabricated Products (3999)	1244	56	5%-10%	62-124	3-6	56-111	1-3
Rubber & Plastic Footwear (3021)	563	29	5%-10%	28-56	2-3	25-50	1-1.
Misc. Plastic Products (3079)	9155	303	5%-10%	450-915	15-30	410-823	10-20
Bottles							
Garden Hose							
Packaging Materials (including coatings)							
Credit Cards							
Medical Products							
TOTAL				1845-2912	75-116	1804-2828	45-70

TOTAL Modular Impact	Production (Billion $)	Employment (Million)
Direct	1.8-2.9	0.1-0 1
All Other	1.8-2.8	* -0.1
	$3.6-5.7	0.1-0.2

* Less than 50,000.

Source: 1972 Census of Manufactures and ADL estimates.

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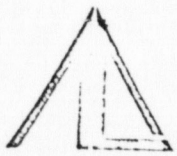

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August 16, 1974

Mr. John H. Stender
Assistant Secretary of Labor
Docket Office, Room 230
OSHA
1726 M Street, N.W.
Washington, D.C. 20210

Dear Mr. Stender:

Thank you for your letter of August 15, 1974, requesting a copy of my presentation at the New York Academy of Sciences meeting on Vinyl Chloride in May, 1974. This paper has just been published in the August, 1974, issue of the Journal of Occupational Medicine and will also appear in the Conference Proceedings of the New York Academy of Sciences.

I am enclosing a copy of my paper; you may use part or all of it for inclusion in your record of the public hearing.

Sincerely,

Irving R. Tabershaw, M.D.

Enclosure

cc: William R. Gaffey, Ph.D.

IRT/ab

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Mortality Study of Workers in the Manufacture of Vinyl Chloride and its Polymers*

Irving R. Tabershaw, M.D. and William R. Gaffey, Ph.D.

Animal studies have shown that inhalation of vinyl chloride produces in rats angiosarcoma of the liver as well as cancers of the lung, kidney, skin and other sites. Although workers in occupations involving exposure to vinyl chloride have been found to have an increased risk of hemangiosarcoma, no excess of other cancers has so far been reported.

This historical prospective mortality study of 8384 men who had at least one year of occupational exposure to vinyl chloride before December 31, 1972, demonstrated that cancers of the digestive system (primarily angiosarcoma), respiratory system, brain, and cancers of unknown site, as well as lymphomas, occurred more often than expected in those members of the study population with the greatest estimated exposure. The mortality from other cancers was lower than that of the general male population, with the exception of cancers of the buccal cavity and pharynx. There was an excess of these cancers, which however was inversely related to estimated exposure. The explanation for the latter finding is not apparent.

The other major findings of the study are: (1) The overall mortality of the study population was approximately 75% of what would be expected in a comparable population of U.S. males; (2) No cause of death showed a statistically significant excess over what would be expected in a comparable U.S. male population; and, (3) No deaths identified as angiosarcoma of the liver were found other than those previously identified.

This is the first epidemiological study which suggests that in humans vinyl chloride may also be associated with cancer of multiple sites.

Drs. Tabershaw and Gaffey are from Tabershaw-Cooper Associates, Inc., Suite 308, 6000 Executive Blvd., Rockville, MD 20852.

*The research on which this report is based was supported by a group of companies engaged in the synthesis and/or the polymerization of vinyl chloride, and administered on their behalf by the Manufacturing Chemists Association.

Vinyl chloride in its manufacture and polymerization has been identified as a narcotizing agent,[1] as a liver toxin,[2,3] and as a vasospastic agent producing a specific occupational disease, acroosteolysis.[4] Recently, vinyl chloride has been incriminated as a carcinogen producing in a group of workers engaged in the manufacture of polyvinyl chloride a rare fatal liver tumor, hemangiosarcoma.[5] Large doses of the chemical in rats reportedly produced cancer of the skin, lung and other organs.[6] Unpublished but public information[7] indicates that inhalation experiments with rats in doses easily reached in manufacturing operations produces in addition to angiosarcoma of the liver, skin, kidney and other malignant lesions.

The present study, however, was not restricted to the conditions and sites suggested by the above investigations, but concerned itself with the entire spectrum of causes of death, to the extent permitted by the size of the study group. The objectives of the study were: (1) To compare the mortality of individuals who have worked in vinyl chloride plants with that of the general population; (2) To compare mortality patterns within the population of vinyl chloride workers, based upon estimated occupational exposure; and (3) To compare mortality among vinyl chloride workers with the mortality of other occupational groups.

The study population consisted of individuals from 33 plants who had worked for at least one year in a job involving exposure to vinyl chloride before December 31, 1972, and included retired and terminated as well as active workers. For each such worker the date of birth and an employment history were obtained, and the vital status of the worker as of December 31, 1972, was ascertained. For those found to have died, those death certificates that were available were obtained and the cause of death determined. The observed mortality was compared with that of the United States male population.

Data Collection

In each plant, data were collected for each worker stated by the plant management to have been employed for at least one year in a job involving exposure to vinyl chloride. In some plants, particularly those producing the monomer, this determination could be made on the basis of job titles. Usually, however, exposure was a function of both job title and the location of the job in the plant, so that the assessment of exposure had to be made on a case-by-case basis by plant officials.

Data were collected for as far back in time as complete records were kept. In most cases this covered the entire history of the plant. In others, records were kept for a fixed period such as a decade. In a few, records were kept for different periods, depending on whether the worker had died on the job or had left employment.

In most plants it was impossible to quantify exposure. However, industrial hygiene and safety personnel in each plant were able to identify certain jobs and locations as involving the highest exposures in the plant, and to classify other exposures as medium or low relative to the "high" represented by the jobs with the greatest exposure. Consequently, each exposed job in a worker's history was scored 1, 2, or 3 to indicate low, medium or high estimated exposure.

This gross classification has two major failings, as a result of the subjective nature of the estimates. The first is that the scores represent estimated relative exposure within a *given* plant. It is therefore possible that, in objective terms, a "high" score in one plant corresponds to a "medium" or even "low" score in another. The second is that the scores usually do not take into account changes in exposure over time. A worker with long service may therefore have had jobs in the remote past which involved "low" exposure relative to other jobs at that time, but which might be "high" in comparison with current exposures in the same job. This subjective classification is therefore of questionable validity in characterizing the exposure of a given worker. For epidemiological purposes, however, those who have high scores can reasonably be expected, on the average, to have had the greatest exposure, while those with low scores will have had the least, even though the true exposure in each group may vary considerably from person to person.

The estimated exposure history of each worker was summarized by calculating an Exposure Index (EI). This was done by multiplying the number of months on each job by the exposure score, totalling these overall exposed jobs, and dividing by the total number of months of exposure.

Follow-up of Study Population

A follow-up procedure was instituted for those who had left employment and whose vital status could not be determined at the local plant, using direct mail follow-up and retail credit bureau investigations. Table 1 shows the vital status of the population as of December 31, 1972. Follow-up is 85% complete. Those who were not found were born (and began their exposure) about ten years before the group on which follow-up was complete, and had about half the duration of employment in exposed jobs with a slightly higher EI. Although there appears to be nothing very unusual about this group in terms of work history and exposure, it is nevertheless true that their exposures took place further back in time than that of the group successfully traced. It is therefore possible that their mortality, after a substantial latent period, might show a somewhat different pattern from that of the traced group.

All of the subsequent analysis is concerned with the 7128 workers on whom follow-up was complete. Table 2 shows their distribution by duration of exposed employment and the year in which that employment began. Although almost half the study group first entered exposed employment in 1960 or later, there are nevertheless 854 workers with 20 years or more exposure, and 1640 with 15 years or more. Table 3 shows the relationship between duration of exposure and EI. There does not appear to be a close relationship between the EI

Table 1. — Follow-up Status of 8384 Vinyl Chloride Workers.

	Total	Alive	Dead	Unknown	Death Certificates	
					Rec'd	Not Rec'd
Number	8384	6776	352	1256	328	24
Percent	100.0	80.8	4.2	15.0	92.7	7.3

and the duration of exposure, that is workers with a higher EI do not differ substantially in duration of exposure from those with a lower EI. One implication is that in assessing the relationship between mortality and exposure, both duration and level of exposure can be examined separately, as well as in combination.

Calculation of Risk of Death

The risk of death is expressed as a Standardized Mortality Ratio (SMR), which is the ratio of the number of observed deaths in the study population to the number of deaths to be expected in a comparable population of U.S. males. SMR's were calculated for overall mortality and for 33 major cause groups.

Table 4 shows observed and expected deaths, and the SMR, for each of these causes for the total study group. In calculating the SMR's for specific causes, the 24 deaths for which no certificates were found were assumed to have the same cause distribution as those for which certificates were available.

In the standard population, each SMR would be equal to 100. Therefore, the statistical significance of the deviation of each SMR in the study population from the expected value of 100 was tested.[8] A single dagger indicates those SMR's which differed significantly from 100 at the 5% level, that is, which had a probability of .05 or less of occurring by chance. A double dagger indicates those which were significant at the 1% level. SMR's based on fewer than five observed cases were not tested for significance.

Table 5 shows the same SMR's for workers with an Exposure Index below 1.5 versus those at 1.5 or above. The dividing point of 1.5 represents a level halfway between "low" and "medium."

Table 6 shows similar results for workers with less than five years exposure versus those with five years or more.

In order to examine the possible interaction between duration and level of exposure, the study population was divided into four groups on the basis of both EI (low vs high) and duration of exposure (short vs long) using the same dichotomization as Tables 5 and 6.

Table 7 shows the results for short versus long exposure in the low EI group, and Table 8 shows the same comparison in the high EI group.

In each of the above tables, deaths for which certificates had not been received were assumed to be distributed as a uniform percentage of all causes. The cause specific SMR's were therefore adjusted upward by a percentage which varied in each subgroup.

Results of Analysis

The overall mortality of the study population is statistically significantly lower than that of the U.S. male population. There were 352 observed deaths compared with 467 expected, for an SMR of 75.

Table 4 shows that no specific cause of death was statistically significantly greater than expected. Several, particularly heart disease, accidents and "other diseases" not detailed in the tables, were significantly below their expected values.

When the study population is divided according to intensity and duration of exposure (Tables 5 and 6) and combinations of these measurements (Tables 7 and 8) three major patterns emerge.

For malignant neoplasms as a whole, the SMR increases with increasing exposure, whether measured by level, duration, or both. In the high exposure group with 5 years or more exposure (Table 8) there are 36 observed cases and 26.11 expected.

For cardiovascular — renal diseases as a group, there are also increases in the SMR with increasing exposure, but the numbers of observed cases remain less than expected, the differences being statistically significant in all groups except the high exposure, long duration group in Table 8.

For all other causes, there are no consistent relationships with exposure.

Within the malignant neoplasms, the largest (although not statistically) significant SMR is in cancers of the buccal cavity and pharynx, with five observed, 2.84 expected, and an SMR of 189. However, Tables 5 to 8 show that all these cases have Exposure Indexes below 1.5 and four out of the five have less than five years exposure. Table 10 is a listing of these deaths with age at death, duration of exposure, and cause as stated on the death certificate.

Cancer of the digestive system shows

Table 2. — Distribution of Months in Exposed Employment by Year in Which Exposure Began, for 7128 Vinyl Chloride Workers with Completed Follow-up.

Year Exp. Started	Total	Months of Exposure							
		< 60	60-119	120-179	180-239	240-299	300-359	360-419	Unknown
1930-39	35	2	4	1	4	6	13	5	
1940-49	1048	135	93	119	151	277	237	34	2
1950-59	1952	389	257	383	631	282			20
1960-69	3368	1714	1442	195					17
1970-71	715	715							
Total	7128	2955	1796	698	786	565	250	39	39

Table 3. — No. and % of 7128 Vinyl Chloride Workers by Months of Exposed Employment and Exposure Index.

Exposure Index	Total	Months of Exposure							
		< 60	60-119	120-179	180-123	240-299	300-359	360-419	Unknown
	No. %	No. %	No. %	No. %	No. %	No. %	No. %	No. %	
1.0-1.4	4032 (100)	1715 (43)	1125 (28)	402 (10)	406 (10)	247 (7)	92 (2)	18 (0)	
1.5+	3057 (100)	1240 (41)	671 (22)	295 (10)	380 (12)	291 (10)	159 (5)	21 (1)	
Unknown	39								39
Total	7128 (100)	2955 (41)	1796 (25)	697 (10)	786 (11)	565 (8)	251 (4)	39 (1)	39 (1)

Table 4. — Observed Deaths Expected Deaths and Standardized Mortality Ratios in Vinyl Chloride Workers.

Cause of Death with I.C.D. Number	Obs/Exp	SMR*
All causes	352/467.28	75 †
Tuberculosis (001-019)	1/5.71	19
Tuberculosis of respiratory system (001-008)	1/5.34	20
Malignant neoplasms (140-205)	29/77.16	110
Malignant neoplasms, buccal cavity and pharynx (140-148)	5/2.84	189
Malignant neoplasms, digestive organs and peritoneum (150-159)	19/21.67	94
Malignant neoplasms, respiratory system (160-164)	25/23.93	112
Malignant neoplasms, genital organs (170-179)	3/3.55	91
Malignant neoplasms, urinary organs (180-181)	1/3.60	30
Malignant neoplasms, other and unspecified sites (190-199)	17/11.75	155
Leukemia and aleukemia (204)	3/3.77	85
Lymphomas (200-203, 205)	6/5.06	106
Diabetes mellitus (260)	7/6.31	120
Major cardiovascular and renal diseases (330-334, 400-468, 592-594)	155/207.46	80 †
Vascular lesions affecting CNS (330-334)	13/24.48	57 †
Rheumatic fever & chronic rheumatic heart dis. (400-402, 410-416)	5/6.87	78
Arteriosclerotic heart disease (420)	121/137.33	95
Nonrheumatic endocarditis (421, 422)	1/6.58	16
Hypertensive heart disease (440-443)	3/9.33	35
Other hypertensive disease (444-447)	3/2.60	124
Chronic & unspecified nephritis & renal sclerosis (592-594)	0/4.27	0
Influenza and pneumonia (480-493)	5/9.96	54 †
Ulcer of stomach and duodenum (540-541)	2/3.83	56
Appendicitis (550-553)	0/0.66	0
Hernia and intestinal obstruction (550-552, 560)	1/1.51	71
Gastritis, duodenitis, enteritis and colitis (558, 571-572)	1/1.31	82
Cirrhosis of liver (581)	3/15.60	21
Hyperplasia of prostate (610)	0/0.39	0
Symptoms, senility and ill-defined conditions (780-795)	1/7.34	15
All other diseases (residual)	21/45.78	49 †
Motor vehicle accidents (810-835)	17/32.70	56 †
Other accidents (800-807, 840-962)	18/30.64	63 †
Suicide (963, 970-979)	16/16.83	102
Homicide (964, 980-985)	1/11.98	9
No. of workers	7128	
Person-years	77846	

*SMR's adjusted for deaths with cause unknown.
†Significant at 5% level.
‡Significant at 1% level.

no excess in the study population as a whole. However, in those workers with an EI of 1.5 or higher, there are 12 observed cases where 9.14 are expected (Table 5). In the subgroup of the above workers with five years or more exposure, there are 11 observed cases and 7.47 expected.

Respiratory cancer shows a slight excess in the total group, and a similar pattern for different exposure categories, with 13 observed versus 10.28 expected when the Exposure Index is 1.5 or higher, and 12 observed versus 8.50 expected when, in addition, the duration of exposure is five years or more.

Malignant neoplasms of other and unspecified sites show an excess in the total group, and an increase with both level and duration of exposure (Tables 5 and 6). The relationship with exposure is more pronounced, since those with exposures of less than five years have fewer cases than expected.

The lymphomas, although occurring at about the expected rate when the whole group is considered, are concentrated almost entirely in the high exposure, long duration group. In that category there are four cases observed and 1.84 expected.

Cancers of the genital and urinary organs, and leukemia, have fewer cases than expected. The number of cases is too small to examine any trends.

Discussion

The favorable overall mortality of the study population is a phenomenon commonly observed in working populations. Standardized Mortality Ratios in the low 80's and below have been found[9, 10]. Even in occupations with well defined hazards which cause an increased risk from a specific cause, the overall mortality may still be favorable because of the low risk from other major causes of death, frequently in the cardiovascular — renal category[11].

Table 5. — Observed Deaths Expected Deaths and Standardized Mortality Ratios in Vinyl Chloride Workers, by Estimated Level of Exposure

Cause of Death with I.C.D. No.	EI < 1.5		EI ≥ 1.5	
	Obs/Exp	SMR*	Obs/Exp	SMR*
All Causes	188/270.33	70 †	157/195.68	80 †
Tuberculosis (001-019)	0/3.38	0	0/2.33	0
Tuberculosis of respiratory system (001-008)	0/3.16	0	0/2.18	0
Malignant neoplasms (140-205)	37/44.28	90	41/32.67	134
Malignant neoplasms, buccal cavity and pharynx (140-149)	5/1.62	330	0/1.21	0
Malignant neoplasms, digestive organs and peritoneum (150-159)	7/12.50	60 †	12/9.14	141
Malignant neoplasms, respiratory system (160-164)	11/13.56	86	13/10.28	135
Malignant neoplasms, genital organs (170-179)	2/7.30	93	1/1.43	75
Malignant neoplasms, urinary organs (180-181)	1/2.07	51	0/1.52	0
Malignant neoplasms, other and unspecified sites (190-199)	9/6.57	146	8/4.52	190
Leukemia and aleukemia (204)	1/2.18	49	2/1.57	136
Lymphomas (200.233, 205)	1/3.48	31	5/2.54	212
Diabetes mellitus (260)	5/3.65	146	2/2.65	81
Major cardiovascular and renal diseases (330-334, 400-468, 592-594)	84/120.11	75 †	69/86.99	85 †
Vascular lesions affecting CNS (330-334)	7/14.42	52 *	6/10.06	64
Rheumatic fever & chronic rheumatic heart dis. (400-402, 410-416)	3/3.98	80	2/2.85	75
Arteriosclerotic heart disease (420)	68/78.94	92	51/58.05	95
Nonrheumatic endocarditis (421, 422)	0/4.20	0	1/2.89	38
Hypertensive heart disease (440-443)	1/5.48	19	2/3.86	56
Other hypertensive disease (444-447)	1/1.52	70	2/1.07	201
Chronic & unspecified nephritis & renal sclerosis (592-594)	0/2.50	0	0/1.77	0
Influenza and pneumonia (480-493)	5/5.80	92	0/4.13	0
Ulcer of stomach and duodenum (540, 541)	1/2.21	48	1/1.60	63
Appendicitis (550-553)	0/0.39	0	0/0.27	0
Hernia and intestinal obstruction (560, 561, 570)	0/0.88	0	1/0.63	171
Gastritis, duodenitis, enteritis and colitis (543, 571, 572)	0/0.76	0	1/0.55	195
Cirrhosis of liver (581)	2/8.90	23	1/6.64	16
Hyperplasia of prostate (610)	0/0.25	0	0/0.14	0
Symptoms, senility and ill-defined conditions (780-795)	0/4.22	0	1/3.09	34
All other diseases (residual)	14/21.80	68 †	6/15.69	41 *
Motor vehicle accidents (810-835)	8/19.08	45 *	9/13.46	72
Other accidents (800-802, 840-962)	11/17.83	66 †	6/12.67	50 *
Suicide (963, 970-979)	9/9.73	98	7/7.02	107
Homicide (964, 980-985)	0/6.98	0	1/4.94	21
No. of workers	4032		3057	
Person-years	45354		32108	

*SMR's adjusted for deaths with cause unknown
†Significant at 5% level
*Significant at 1% level

In view of these facts, SMR's which are higher than expected may be worthy of attention even if they are not statistically significant. This is especially true in the present study since the number of deaths from many causes is quite small, and even a relatively high SMR may not reach statistical significance.

If, in addition, a particular cause shows a consistent pattern of increase with exposure or estimated exposure, the findings are particularly interesting.

By these criteria, mortality from digestive cancer, respiratory cancer, cancer of other and unspecified sites, and lymphomas, appear to be related to exposure as defined in this study.

In view of the association between vinyl chloride exposure and angiosarcoma of the liver, the digestive cancers were examined further to see what contribution angiosarcoma made to the observed mortality pattern.

Of the 19 digestive cancers, seven were liver cancers, of which two were angiosarcomas according to the death certificate. However, among angiosarcoma deaths in vinyl chloride workers identified by other investigators, there were six which occurred in the present study population during the study period. They were all found in the course of the study. Table 11 shows these cases with the cause of death as given on the death certificate. Note that one case was certified as cirrhosis, and was so considered throughout this study, since the validity of comparisons with population data required that cause of death be determined only from information on the death certificate. The other five were correctly classified as

Table 6. — Observed Deaths:Expected Deaths and Standardized Mortality Ratios in Vinyl Chloride Workers by Duration of Exposed Employment.

Cause of Death with I.C.D. No.	< 60 months		≥ 60 months	
	Obs:Exp	SMR*	Obs:Exp	SMR*
All causes	94:140.53	67 †	251:329.30	76 †
Tuberculosis (001-019)	0/2.23	0	0/3.55	0
Tuberculosis of respiratory system (001-008)	0/2.07	0	0/3.33	0
Malignant neoplasms (140-205)	13/19.96	78	65/57.61	116
Malignant neoplasms, buccal cavity and pharynx (140-148)	4/0.70	688	1/2.16	47
Malignant neoplasms, digestive organs and peritoneum (150-159)	2/5.26	46	17/16.56	106
Malignant neoplasms, respiratory system (160-164)	3/5.52	65	21/18.51	116
Malignant neoplasms, genital organs (170-179)	0/0.99	0	3/2.76	112
Malignant neoplasms, urinary organs (180-181)	0/0.83	0	1/2.79	37
Malignant neoplasms, other and unspecified sites (190-199)	2/3.40	71	15/8.23	187
Leukemia and aleukemia (204)	1/1.25	96	2/2.53	81
Lymphomas (200.203, 205)	1/2.01	60	5/4.07	125
Diabetes mellitus (260)	2/1.80	134	5/4.54	117
Major cardiovascular and renal diseases (330-334, 400-468, 592-594)	28/51.45	65 ‡	125/157.39	81 ‡
Vascular lesions affecting CNS (330-334)	4/5.97	81	9/18.71	49 ‡
Rheumatic fever & chronic rheumatic heart dis. (400-402, 410-416)	2/2.39	101	3/4.53	68
Arteriosclerotic heart disease (420)	21/32.54	78 †	98/105.39	96
Nonrheumatic endocarditis (421, 422)	0/1.79	0	1/5.35	20
Hypertensive heart disease (440-443)	1/2.42	49	2/7.01	30
Other hypertensive disease (444-447)	0/0.79	0	3/1.82	170
Chronic & unspecified nephritis & renal sclerosis (592-594)	0/1.55	0	0/2.76	0
Influenza and pneumonia (480-493)	3/2.93	123	2/7.09	29
Ulcer of stomach and duodenum (531-541)	1/1.07	112	1/2.78	37
Appendicitis (550-553)	0/0.24	0	0/0.43	0
Hernia and intestinal obstruction (560, 561, 570)	0/0.42	0	1/1.10	93
Gastritis, duodenitis, enteritis and colitis (543, 571, 572)	1/0.40	301	0/0.92	0
Cirrhosis of liver (581)	1/4.49	26	2/11.18	18
Hyperplasia of prostate (610)	0/0.07	0	0/0.33	0
Symptoms, senility and ill-defined conditions (780-795)	1/2.28	53	0/5.09	0
All other diseases (residual)	4/11.97	40	16/26.34	63 †
Motor vehicle accidents (810-835)	10/16.14	75	7/16.65	43 ‡
Other accidents (830-802, 840-962)	7/12.94	65 †	10/17.82	58 †
Suicide (963, 970-979)	6/6.34	114	10/10.28	100
Homicide (964, 980-985)	1/5.80	20	0/6.20	0
No. of Workers	2955		4134	
Person-years	34201		43240	

*SMR's adjusted for deaths with cause unknown
†Significant at 5% level
‡Significant at 1% level

Table 7. — Observed Deaths/Expected Deaths and Standardized Mortality Ratios in Vinyl Chloride Workers with Exposure Indices Below 1.5, by Duration of Exposed Employment.

Cause of Death with I.C.D. No.	< 60 Months Exposure		≥ 60 Months Exposure	
	Obs/Exp	SMR*	Obs/Exp	SMR*
All Causes	56/89.23	63‡	132/181.28	73‡
Tuberculosis (001-019)	0/1.41	0	0/1.97	0
Tuberculosis of respiratory system (001-008)	0/1.31	0	0/1.85	0
Malignant neoplasms (140-205)	8/12.86	73	29/31.46	95
Malignant neoplasms: buccal cavity and pharynx (140-148)	4/0.45	1036	4/1.17	88
Malignant neoplasms: digestive organs and peritoneum (150-159)	1/3.43	34	6/9.08	68
Malignant neoplasms: respiratory system (160-164)	2/3.58	65	9/10.00	93
Malignant neoplasms: genital organs (170-179)	0/0.68	0	2/1.62	127
Malignant neoplasms: urinary organs (180-189)	0/0.55	0	1/1.53	67
Malignant neoplasms: other and unspecified sites (190-199)	1/0.97	120	8/4.43	187
Leukemia and aleukemia (204)	0/0.79	0	1/1.40	73
Lymphomas (200-203, 205)	0/1.26	0	1/2.23	46
Diabetes mellitus (260)	2/1.15	203	3/2.59	124
Major cardiovascular and renal diseases (330-334, 400-468, 592-594)	21/33.38	73 ‡	63/85.82	75 ‡
Vascular lesions affecting CNS (330-334)	2/3.93	59	5/10.51	49 †
Rheumatic fever & chronic rheumatic heart dis. (400-402, 410-416)	2/1.50	155	1/2.49	41
Arteriosclerotic heart disease (420)	16/21.14	88	52/57.87	93
Nonrheumatic endocarditis (421-422)	0/1.18	0	0/3.02	0
Hypertensive heart disease (440-443)	1/1.58	74	0/3.90	0
Other hypertensive disease (444-447)	0/0.50	0	1/1.02	101
Chronic & unspecified nephritis & renal sclerosis (592-594)	0/0.97	0	0/1.53	0
Influenza and pneumonia (500-493)	3/1.86	189	2/3.95	53
Ulcer of stomach and duodenum (540-541)	0/0.68	0	1/1.53	67
Appendicitis (550-553)	0/0.15	0	0/0.24	0
Hernia and intestinal obstruction (560, 561, 570)	0/0.27	0	0/0.61	0
Gastritis, duodenitis, enteritis and colitis (543, 571, 572)	0/0.26	0	0/0.51	0
Cirrhosis of liver (581)	1/2.80	42	1/6.09	16
Hyperplasia of prostate (600)	0/0.05	0	0/0.20	0
Symptoms, senility and ill-defined conditions (780-795)	0/1.43	0	0/2.79	0
All other diseases (residual)	4/7.45	63	10/12.92	79
Motor vehicle accidents (810-835)	3/9.87	35	5/9.22	56
Other accidents (800-802, 840-962)	3/7.98	44	8/9.85	83
Suicide (953, 970-979)	3/4.08	80	6/5.55	109
Homicide (964, 960-965)	0/3.55	0	0/3.41	0
No. of Workers	1715		2317	
Person-years	21418		23970	

*SMR's adjusted for deaths with cause unknown
‡Significant at 5% level
†Significant at 1% level

Table 8. — Observed Deaths Expected Deaths and Standardized Mortality Ratios in Vinyl Chloride Workers with Exposure Indices of 1.5 or Greater, by Duration of Exposed Employment

Cause of Death with I.C.D. No.	< 60 Months Exposure		≥ 60 Months Exposure	
	Obs/Exp	SMR*	Obs/Exp	SMR*
All Causes	38/47.93	79	119/147.81	81 †
Tuberculosis (001-019)	0/0.76	0	0/1.57	0
Tuberculosis of respiratory system (001-008)	0/0.71	0	0/1.48	0
Malignant neoplasms (140-205)	5/6.57	96	36/26.11	141
Malignant neoplasms, buccal cavity and pharynx (140-148)	0/0.23	0	0/0.99	0
Malignant neoplasms, digestive organs and peritoneum (150-159)	1/1.67	76	11/7.47	151
Malignant neoplasms, respiratory system (160-164)	1/1.79	71	12/8.50	144
Malignant neoplasms, genital organs (170-179)	0/0.29	0	1/1.41	73
Malignant neoplasms, urinary organs (180-181)	0/0.26	0	0/1.26	0
Malignant neoplasms, other and unspecified sites (190-199)	1/1.18	107	7/3.51	204
Leukemia and aleukemia (204)	1/0.44	288	1/1.13	90
Lymphomas (200.203, 205)	1/0.71	178	4/1.84	222
Diabetes mellitus (260)	0/0.61	0	2/2.04	100
Major cardiovascular and renal diseases (330-334, 400-468, 592-594)	7/16.54	54 ‡	62/70.45	90
Vascular lesions affecting CNS (330-334)	2/1.87	135	4/8.19	50
Rheumatic fever & chronic rheumatic heart dis. (400-402, 410-416)	0/0.82	0	2/2.04	100
Arteriosclerotic heart disease (420)	5/10.41	61 †	45/47.65	98
Nonrheumatic endocarditis (421, 422)	0/0.57	0	1/2.32	44
Hypertensive heart disease (410-443)	0/0.76	0	2/3.10	66
Other hypertensive disease (434-447)	0/0.27	0	2/0.81	253
Chronic & unspecified nephritis & renal sclerosis (592-594)	0/0.54	0	0/1.23	0
Influenza and pneumonia (490-493)	0/0.93	0	0/3.13	0
Ulcer of stomach and duodenum (530, 541)	1/0.35	362	0/1.25	0
Appendicitis (550-553)	0/0.08	0	0/0.35	0
Hernia and intestinal obstruction (560, 561, 570)	0/0.14	0	1/0.49	209
Gastritis, duodenitis, enteritis and colitis (543, 571, 572)	1/0.14	904	0/0.41	0
Cirrhosis of liver (581)	0/1.56	0	1/5.08	20
Hyperplasia of prostate (212)	0/0.01	0	0/0.13	0
Symptoms, senility and ill-defined conditions (780-795)	1/0.80	158	0/2.30	0
All other diseases (residual)	0/4.02	0	6/11.88	51 *
Motor vehicle accidents (810-835)	7/5.05	146	2/7.43	28
Other accidents (810-832, 840-922)	4/4.73	107	2/7.96	26
Suicide (953, 970-979)	3/2.40	158	4/4.62	88
Homicide (964, 965-969)	1/2.18	58	0/2.76	0
No. of Workers	1240		1817	
Person-Years	12828		19305	

*SMR's adjusted for deaths with cause unknown
†Significant at 5% level
‡Significant at 1% level

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Table 9. — Other Malignancies (190-199, I.C.D.) in VDM Epidemiology Study Population.

Study No.	Age at Death	Mos. Exposed	Cause as Given	Autopsy
2744	38	186	Widespread metastatic melanoma (1 yr.) Malignant melanoma of back	yes
3700	48	50	Malignant melanoma with widespread metastases	no
2096	67	69	Brain tumor (carcinoma) (8 mos.)	
3946	43	64	Meningitis and pneumonitis (5 wks.) Ependymoma, fourth ventricle brain (2 mos. post-op craniotomy)	no
4433	54	81	Astrocytoma, malignant left cerebral hemisphere (1 yr.)	yes
4800	61	51	Carcinoma of the brain Arteriosclerosis Pneumonia due to static congestion	no
7279	57	271	Brain tumor, glioblastoma multiforme (18 mos.)	
7300	44	211	Brain tumor, malignant (2 mos.)	
7371	56	249	Brain tumor, malignant	no
7306	53	216	Thyroid carcinoma with metastases	no
5769	52	239	Carcinomatosis Carcinoma vertebral body	yes
5245	67	201	Liposarcoma with metastasis (3 mos.)	yes
2850	64	193	Carcinomatosis, primary region not known (Past history, rheumatoid arthritis, myocardial infarction)	no
4778	54	300	Cardiac arrest (1 hr.) Respiratory arrest and cerebral hypoxia (2 days) Widespread metastatic carcinoma (2 mos.)	
4786	61	318	Generalized metastasis undifferentiated (7 mos.) Squamous cell carcinoma, primary site undetermined	yes
5783	69	166	Metastatic carcinoma of abdomen Critical site undetermined (Pulmonary emphysema)	
7301	55	133	Carcinomatosis (4 mos.) Primary site undetermined	no

liver cancer, but only two were specified as angiosarcoma.

If there had been no angiosarcomas, the number of deaths classified as cirrhosis in this study would have decreased by one, and the number classified as digestive cancer would have decreased by five. The pattern of duration and intensity of exposure in these five cases was such that if they had not been present there would have been no relationship between exposure and digestive cancer. The mortality pattern in this cause group is therefore attributable to angiosarcomas of the liver.

The other cause group worth further investigation is cancer of other and unspecified sites, both because it is a heterogeneous category and because it seems, unlike the other cancers, to be more related to duration than to level of exposures.

Table 9 shows a list of the specific causes included in this category, which is essentially brain cancer and generalized cancer with primary site unknown. About 40% of the observed deaths were due to brain cancer. In the general male population, about 22% of this category is due to brain cancer, so that not only is the mortality from cancer of other and unspecified sites excessive, but brain cancer is overrepresented within the category.

The possibility exists based on the lack of specificity of some of the listed causes that some of the brain cancers were not primary, but metastases from another unidentified site such as the lung.

The cancers of the buccal cavity and pharynx are difficult to explain because of their occurrence in the low exposure, short exposure group. It is possible that this is a chance occurrence, that exposures to other substances were involved, or that the mouth and pharynx may be peculiarly susceptible because of the gaseous nature of the chemical.

Possible Biases in the Calculation of Risk

There are two major potential sources of bias in the study. The first is that the follow-up rate is lower than is desirable. The second is that observations of workers with long exposures followed by a long latent period are not adequately represented, so that the power of the study to detect causes of death associated with long exposure and long latency is impaired. Populations by such characteristics exist and should be investigated.

Table 10. — Malignant Neoplasms of Buccal Cavity and Pharynx (140-148, I.C.D.) in VCM Epidemiology Study Population.

Study No.	Age at Death	Mos. Exposed	Cause as Given	Autopsy
3003	31	49	Carcinoma of lip with metastasis to lung and neck (5 yrs.)	no
3431	56	30	Pulmonary edema (50 min.) Pulmonary metastases (Carcinoma epidermoid, tongue and mandible)	no
3001	57	71	Metastatic squamous cell ca. primary lesion palate	yes
7365	54	40	Atelectasis due to metastasis to mediastinum (4 mos.) Multiple malignant metastases to brain, liver & mediastinum (1 yr.) Adeno carcinoma of nasal pharynx (2 yrs.)	yes
3354	63	26	Massive hemorrhage into tracheo tree Status post laryngo pharyngectomy, left radial neck dissection Well differentiated keratinizing squamous cell carcinoma of left pyriform sinus (mos.)	yes

Table 11. — Angiosarcoma Deaths in VCM Epidemiology Study Population.

Study No.	Age at Death	Mos. Exposed	Cause as Given	Autopsy
4250	54	203	Cirrhosis of liver (sev. weeks)	yes
4255	60	281	Bleeding from hepatoma (3 days) (Laennecs cirrhosis)	yes
5765	45	167	Angiosarcoma rt. lobe of liver (10 mos.)	yes
7203	38	174	Liver failure (1 mo.) Cancer of liver, primary (15 mos.)	
7376	52	238	Cardiac tampanade and massive left hemothorax (mins.) Widely metastatic angiosarcoma of liver (4 mos.)	
7385	43	214	Hepatic failure Primary carcinoma liver	yes

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W. O. Baker
President

August 15, 1974

Docket Office OSH-36
Committee Management Office
Occupational Safety and Health Administration
Room 200
1726 M Street N.W.
Washington, D.C. 20210

Gentlemen:

The Bell System relies heavily upon poly (vinyl chloride) as an insulation and jacketing material for wire and cable used inside of telephone offices and on customer premises. In 1974, the Bell System will use about 50 million pounds of PVC in wires, cables and cords. The unique flame retardant character of PVC was a crucial factor in its choice for these applications. No suitable substitute material is known, and we do not anticipate that one will be developed in the near future. Polyolefins are the only plastics produced in comparable volume, and they are highly flammable and less durable in many applications.

PVC is used in the Bell System in a wide variety of ways. Its combination of inherent flame retardance, resistance to wear, good electrical properties, and long life make it an extremely valuable material. Very large quantities are used in wire insulation, conduit, and central office components and there is no known satisfactory replacement for it in these applications. Although other materials can be used in place of PVC in certain applications, it should be kept in mind that these other materials (e.g. nylon) are also in short supply with no prospect of satisfying the demand that would be created by the loss of PVC. In addition, PVC is unique. Substitutions are made at a sacrifice in important properties and flammability is most significant in this regard. The Bell System has taken the view that flammable cables must not be placed in business locations or in customers' homes. It would be impossible to adhere to this principle if PVC were unavailable.

It is, therefore, apparent that if the supply of PVC were to be cut off the nation's telecommunications system would be severely crippled. Many other telecommunication industries would be similarly affected in greater or lesser degrees.

The discovery of indications that vinyl chloride monomer (VCM) is a carcinogenic agent has created major problems for PVC producers. There is evidence that the producers are responding actively to the elimination of this health hazard. The Bell System does not use VCM in any form and cannot comment on the hazards or the difficulty PVC producers face in reducing exposure. It is our belief, however, that some solution to the problem must be found short of closing off production.

Sincerely yours,

W. O. Baker

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PLEASE REPLY TO: SOUTHBRIDGE PLASTICS DIVISION

August 16, 1974

Occupational Safety & Health Administration
U.S. Department of Labor
Room 200
1726 M Street N.W.
Washington, D.C. 20210

Attention: Docket Officer
Reference: Docket OSH-36

Gentlemen:

We wish to submit the following statement for the post hearing record on the "Proposed Standard for Occupational Exposure to Vinyl Chloride".

The Hatco Plastics Division of W. R. Grace & Co. manufactures a broad range of flexible film, sheeting, coated fabrics and compounds derived from Polyvinyl Chloride (PVC) resins. The principal products include unsupported film and sheeting for use in window shades, shower curtains, ceiling tile, tapes, medical grade films used in the storage of blood and other biological solutions; supported sheeting or vinyl coated fabrics for upholstery, wall coverings, handbag, shoe and other applications; and vinly compounds for use in medical and surgical tubing, wire and cable, shoe, and other applications by the extrusion and injection molding processes.

These materials are manufactured at three plants located at Brooklyn, New York; Corinth, Mississippi; and Los Angeles, California; which employ a total of approximately 1,130 people. Sales in 1973 were in excess of $55 million based on an investment in excess of $30 million.

The principal raw material for all of the Hatco Plastics Division's products is PVC.

We have been informed by our suppliers of PVC resins that the proposed "non-detectable" level of VCM exposure standard is not technologically feasible and its adoption could result in the industry being forced to cease production.

The extent to which other materials could be substituted for PVC resins is negligible in the near term and for most product lines no adequate substitute material exists. Thus, the consequence of a total discontinuance or a sharp reduction in the supply of PVC resins would constitute a disaster in both human and economic terms.

Of particular consequence would be the discontinuance of medical devices derived from PVC. These include blood bags, solution containers, intravenous feeding tubes, urethral and cardiovascular catheters and dialysis equipment - products all vital to patient care. The Food and Drug Administration recently has been investigating possible danger to patients from VCM through the use of finished medical products made from PVC. To our knowledge, their investigation to date have found no detectable levels of VCM present in such products.

At the present state of technology, there are no alternate materials for the production of most of these essential medical devices, nor in most cases, are there acceptable alternate devices or products.

We are in agreement with the SPI and industry position that the proposed "non-detectable" level of VCM exposure standard is unnecessarily harsh and restrictive, and fully support their proposed stepdown time table for VCM exposure levels in the VCM-PVC industry.

We further support the contention that PVC processors and fabricators be exempt from the regulations of the proposed standard by virtue of the fact that the VCM emissions in these industries are either "non-detectable" or of a very low level which can be further reduced by ventilation changes or improved work practices.

Air monitoring programs were initiated at our manufacturing plants even in advance of the April 5th Emergency Temporary Standard for Exposure to Vinyl Chloride.

We are presently in complete compliance with this standard at all our manufacturing facilities. Approximately 25 major operator locations at each plant have been sampled seven times each by OSHA's recommended personnel monitoring method employing carbon absorption tubes, followed by analysis using Gas Chromatography with flame ionization detection.

The results obtained indicate that approximately 98% of some 500 individual sampling performed showed readings of less than 10 ppm of VCM, with approximately 90% of the readings between the "non-detectable" and 1 ppm levels.

In summary, we urgently request that the Occupational Safety and Health Administration adopt a meaningful and resonable VCM exposure standard which will assure the safety of all employees as well as the continued existence of the vital PVC and associated industries.

Respectfully yours,

Laurence Kogos
President
Hatco Plastics Division
W. R. Grace & Co.

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Supplemental Appendix

CHEMICAL CARCINOGENS IN THE ENVIRONMENT AND IN THE HUMAN DIET: CAN A THRESHOLD BE ESTABLISHED?

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In the current atmosphere of concern over the possible deleterious effects of chemicals in the environment to man, one encounters with increasing frequency the claim that, for certain compounds, there is no threshold level of exposure below which no harmful effects on an organism will occur. Such views can be rather easily demonstrated to be toxicologically unsound under experimental conditions when acute and subacute toxicities are in question, since for these, clearcut dose-response curves can be established for the dose levels tested in a variety of test animals. On the other hand, when argument centers around the induction of cancer by chemicals, the problem of defining a "no-effect" exposure level becomes more complex and controversial.

In testing for long-term effects such as carcinogenicity, dose-response curves can, of course, also be constructed. Disagreements in this area of research begin over the meaning of the lower part of the curve and specifically over whether it is

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*/ This text incorporates the major points of a lecture delivered by the senior author at a special seminar in the Department of Pathology, V.A. Hospital and Baylor College of Medicine, Houston, Texas, on December 6, 1973.

feasible to determine a dose which would show no effect in a very large human population on the basis of laboratory studies with a limited number of test animals.

The nature of the problem is as follows. In carcinogenicity experiments, if one tests a particular substance at several dose levels, e.g. using 100 test animals for each dose, positive results can be expressed in a dose-response curve with each point representing the percentage of test animals which developed malignancies. However, even if one knows that at a given dose cancer was induced in 50 percent of the animals, at a lower dose only in 10 percent, and at a still lower one in none, the lowest dose cannot be regarded as a "no-effect" threshold for induction of cancer in whole populations, since the results indicate only that none of the 100 test animals developed cancer at that dose rate. The argument can proceed: if it has been shown that a substance can act as a carcinogen in test animals at some level, and if one assumes that animal experiments can be extrapolated to humans, then one must seek a way of determining the lowest dose which would be safe for--not 100 people--but at least 100,000, or, in the more conservative view, 100 million. Although there have been proposals to initiate experiments utilizing thousands or millions of test animals, most researchers recognize this as impracticable. As stated by Weil (1972, 1973) even if dosing thousands of animals at low dose levels could establish the most probable shape or slope of

a dose-response curve, this slope would have its large uncertainty and would hold only for the specific experimental conditions tested. Even if a low-risk, no-cancer level was found for a chemical under a particular set of conditions, the need to extrapolate from mouse to man still remains. It is largely because of these problems with numbers and with long induction time of effects that the dose-response testing procedures of classical toxicology have been called "crude and insensitive" by some investigators engaged in cancer research, when applied to carcinogenicity testing of chemicals.

Three Proposed Methods for Establishing a "Safe" Exposure Level.

To circumvent the apparent inability to determine through experiment the possible effects on a very large population which might ensue from regular exposure to exceedingly small quantities of a suspected carcinogen, it has been suggested that once a dose-response curve has been obtained for the carcinogenic potential of a chemical in the exposure range where effects are observable, the dose which should produce only one tumor in a population of 100,000, 1 million, or 100 million individuals could be calculated by downward mathematical extrapolations from the observable range. However, such methods have been rejected by some as unreliable. The reasons for the doubtful validity of mathematically defined "safe doses" are elaborated in "The Report of the Carcinogenicity Panel" (in: Report of the Secretary's Commission on Pesticides, 1969;

hereafter referred to as the Mrak Report), whose members cite materials from "The Report of the Subcommittee on Carcinogenesis of FDA Committee on Protocols for Safety Evaluation" (1969), and announce that "the concerned statisticians at the National Cancer Institute concur" with the skepticism expressed in the FDA report. They write: "It might be thought that the basis for such extrapolation could be provided by observations in the observable range. To show how far from being the case this actually is, we give below three diferrent dose-response curves, mathematically defined over a dosage range of 256 fold. All three have the same TD_{50} [dose which results in tumors in 50 percent of the animals] and TD_{16} [tumor does for 16 percent of the animals]. The first is a probit curve, the second a logistic curve, and the third the so-called one-particle curve." (Mrak Report, 1969, p. 493)

A recompilation of the two tables from the Mrak Report illustrating these three curves is presented in Table 1. Since the numbers listed under "Dose" are strictly abstract proportions, "1" stands for the dose which would produce tumors in 50 percent of the test animals, and all the other numbers represent multiples or fractions of that dose. The authors comment: "It will be noted that below the TD_{50} the three curves differ by little and that in any experiment of practicable size (say less than several thousand animals) it would not be possible to conclude from the actual observations which one of the three best described the data. As shown below, however, the $TD_{.0001}$

TABLE 1

RECOMPILATION OF TABLES FROM THE
"REPORT OF THE CARCINOGENICITY PANEL"
(AFTER MRAK REPORT, 1969, p.494)

TD (%) on Probit Curve	Abstract Dose	TD (%) on Logistic Curve	Abstract Dose	TD (%) on One-Particle Curve	Abstract Dose
98.0	16.0	96.0	16.0	100.0	16.0
93.0	8.0	92.0	8.0	99.0+	8.0
84.0	4.0	84.0	4.0	94.0	4.0
69.0	2.0	70.0	2.0	75.0	2.0
50.0	1.0	50.0	1.0	50.0	1.0
31.0	0.5	30.0	0.5	29.0	0.5
16.0	0.25	16.0	0.25	16.0	0.25
7.0	0.125	8.0	0.125	8.0	0.125
2.0	0.062	4.0	0.062	4.0	0.062
1.0	0.04	1.0	0.022	1.0	0.0144
0.1	0.0155	0.1	0.00315	0.1	0.00144
0.001	0.00279	0.001	0.000063	0.001	0.0000144
0.0001	0.00136	0.0001	0.0000098	0.0001	0.00000144
0.000001	0.000412	0.000001	0.00000016	0.000001	0.0000000144

(one in a million dose) and the $TD_{.000001}$ (one in a hundred million dose) obtained by extrapolation of these three curves differ markedly. . . The one in one-hundred million dose, which Mantel and Bryan call the 'virtually' safe dose is one-hundredth the TD_1 using the probit curve, one-hundred thousandth using the logistic and one one-millionth using the one-particle curve." (ibid, p. 494)

The members of the Carcinogenicity Panel and the FDA statisticians further state that from data in the observable range, it is not possible to decide which of the three curves most accurately represents experimental findings, and that extrapolation downward to a "virtually safe dose" is therefore not reliable. The position of both publications is summarized in the following: "Clearly extrapolation from the observable range to a safe dose has many of the perplexities and imponderables of extrapolation from animal to man and it would be imprudent to place excessive reliance on mathematical sleight of hand, particularly when the dose-response curves used are largely empirical descriptions, lacking any theoretical physical or chemical basis." (ibid, p. 495)

Variations on this kind of argument have been presented for the last 15 years or so by proponents of the "no safe dose for carcinogens" school of thought. In 1970, for instance, in the "Report of the Ad Hoc Committee on the Evaluation of Low Levels of Environmental Chemical Carcinogens", the following

claim is several times repeated: "No level of exposure to a chemical carcinogen should be considered toxicologically insignificant for man. For carcinogenic agents a 'safe level for man' cannot be established by application of our present knowledge. The concept of 'socially acceptable risk' represents a more realistic notion." (Ad Hoc Committee, 1970, p. 1)

And further: "In order to evaluate the hazard of a chemical for man, one must extrapolate from the animal evidence. It is essential to recognize that no level of exposure to a carcinogenic substance, however low it may be, can be established to be a 'safe level' for man. This concept, put forward in the 1950's, remains true in 1970." (ibid, p. 4) Finally: "It is impossible to establish any absolutely safe level of exposure to a carcinogen for man. . .. The principle of zero tolerance should be applied in all but the most extraordinary of cases." (ibid, p. 7)

On the face of things, the agnostic stance taken by the writers quoted would seem to rest on genuine "unknowables", and the general conclusion that the only sure way to protect the public is through establishing zero tolerances would appear to express an attitude of prudence in the best tradition of preventive medicine. However, most of these arguments ignore the fact that in order to produce an effect, any material has to be present in a minimal number of atoms, molecules, subatomic particles, or photons. (Dinman, 1972; Rossi and Kellerer, 1972)

In the case of chemicals, the probability that one or even several hundred molecules of a compound will have a deleterious effect on an organism is virtually zero.

The Threhold Concept Applied to Human Intake of DDT.

In order to give concrete meaning to the figures resulting from the differing downward extrapolations presented in Table 1, one has to consider four parameters: (1) A supposedly known carcinogen for which the TD_{50} has been established; (2) the number of molecules present in the T[illegible]se of the compound in question; (3) the number of cel[illegible]sent in the organism for which the "virtually safe dose" or the "acceptable risk dose" needs to be established; and (4) the minimum number of molecules per cell, the presence of which will be registered by that cell.

It has been claimed recently that DDT is an hepatocarcinogen, and, according to the "Bionetics Report (Innes, et al., 1969), the TD_{50} of DDT in mice is about 140 ppm in food. We propose to use DDT as an example for downward extrapolation, with 140 ppm in food as parameter #1, even though we entertain serious doubts that it has actually been demonstrated to be carcinogenic.

Parameter #2 is easily established. On the basis of Avogadro's law, it is known that 1 g of DDT contains 1.7×10^{21} molecules.

Next one should consider how many cells there are in the human body. Different types of cells vary considerably in size, but their weight can be calculated. Dinman (1972), for instance, came to the conclusion that a typical liver cell weighs 7×10^{-9} g. From his data, however, one gathers that he assumed a spherical cell shape. Using a more realistic cylindrical form, one arrives at half of his value: 3.5×10^{-9} g for the average liver cell. A red blood cell, which is of course much smaller, weighs only 9×10^{-11} g; and there are approximately 3×10^{13} red blood cells alone in the 6 liters of blood in the human body. The weight of an average body cell should be very close to 5×10^{-10} g, and if the weight of an average man is expressed as 7×10^{4} g and divided by the average cell weight, the result is 1.4×10^{14} total cells. Different approaches to estimating the number of cells in the human body lead to almost the same figure, so we take this to be a reasonable approximation.

The fourth question--that is, how many molecules are needed in order merely to be registered*/ by a cell--can be answered by considering the following. In 1964, Hutchinson compiled data regarding the numbers of different atoms in one liver

*/ It should be noted that this term has nothing to do with an effect which can be measured, deleterious or otherwise. It is slightly awkward, but we wanted to avoid such anthropomorphic alternatives as "noticed by the cell", which conveys the meaning with more linguistic smoothness but has the disadvantage of implying that there is some kind of conscious recognition involved.

cell. From his tabulation it became evident that those atoms which have a biologically demonstrable function are present in quantities of at least 10^6 to 10^8 per cell. From these considerations he concluded: ". . . there might be too many commoner, accidentally and potentially interfering materials (or sites?) around in the cell for any very important substance to work practically at a concentration of less than 10^4 atoms or molecules per cell" (Hutchinson, 1964, p. 931).

A similar view was expressed by Dinman in 1972. Using both Hutchinson's approach and findings from enzyme inhibition studies with potent inhibitors, Dinman showed that about 4 x 10^4 molecules per cell are required to produce even a minimal degree of inhibition. He wrote: "to believe that a single molecule's presence in a cell implies a definite potential for deleterious effect disregards stochastic considerations. . .. While the construction of stochastically sound model is remote, the reasonableness of the hierarchy of cellular element concentrations as these relate to metabolic function suggest that a threshold for biological activity exists within a cell at 10^4 atoms. . .. The cellular organism operates within a quantitative rate limit that transcends any statements having only qualitative bases. Thus concepts concerning encroachment on response capabilities over a life span are inadequate descriptors of biological activity in the absence of quantitative qualifiers" (Dinman, 1972, p. 497).

Some reservations might be raised about Dinman's generalizations if consideration is given to certain compounds which are in constant circulation, are target specific, and undergo little metabolic transformation or excretion. Uptake of minute quantities of some of these oligodynamic substances is adequate to maintain homeostasis of an organism. In order to examine whether or not a cellular threshold exists at circa 4×10^4 molecules for such chemicals, we selected vitamin B_{12} as an example, using data from Goodman and Gilman (1970). For the purpose of illustration we shall relate molecular quantities of the vitamin to numbers of mature erythrocytes, although it is recognized that B_{12} primarily affects the hemopoetic system. The underlying assumptions and values for all the calculations in the text are summarized in Table 2.

The average daily intake of vitamin B_{12} in man is about 1 ug, which contains 4.5×10^{14} molecules, or only 3 molecules per body cell. However, this quantity is added to an already existent vitamin pool in the body, which is about 5 mg (2.3×10^{18} molecules), half of it in the circulation. The small amounts of vitamin B_{12} present in the daily diet can be taken up by man because it immediately complexes with intrinsic factor; thus at the site of uptake in the ileum, concentrations of about 1.3×10^{15} molecules are present. On the other hand, if the quantity of vitamin B_{12} in the blood falls below 100 pg/ml (2.7×10^{17} molecules in total circulation or 9×10^3 molecules per red corpuscle), as is the case in pernicious anemia, the therapeutic doses recommended

TABLE 2

SUMMARY OF VALUES AND ASSUMPTIONS

Avogadro's number	6.02×10^{23} molecules
Molecular weight, DDT	354.5
Molecular weight, vit. B_{12}	1,355.4
Molecular weight, DAB*	225.3
Molecular weight, MC*	268.3
Molecular weight, AB*	197.2
Weight of liver cell	3.5×10^{-9} g
Weight of erythrocyte	9×10^{-11} g
Weight of average body cell	5×10^{-10} g
In the human liver there are	5×10^{11} cells
In human blood there are	3×10^{13} cells
Weight of average man	7×10^{4} g
In the human body there are	1.4×10^{14} cells
Weight of rat	2.0×10^{2} g
In the rat liver there are	1.4×10^{9} cells
In the rat body there are	4.0×10^{11} cells
Weight of mouse	5.0×10^{1} g
In the mouse liver there are	3.5×10^{8} cells
In the mouse body there are	1.0×10^{11} cells

* See text for full chemical designations

to restore normal circulating levels are from 30 to 100 ug/day, intramuscularly. This amounts to between 500 and 2,000 molecules per erythrocyte, and since excretion of the vitamin is minimal (< 1 ug/day) within a few days such administration should bring the blood level above 10^4 molecules per erythrocyte. One aspect of these figures is noteworthy, i.e., there are only 4×10^4 molecules of the vitamin for each erythrocyte in the normal blood--a figure exactly the same as Dinman's value at which inhibitory molecules can produce minimal enzyme inhibition. Furthermore, if the number of vitamin B_{12} molecules per red blood cell falls by less than one order of magnitude (9×10^3), pernicious anemia generally develops.

From the above considerations, about 4×10^4 molecules per cell seems to be a reasonable figure for parameter #4. We may now proceed to a consideration of the absolute quantities of DDT involved in the two safest exposure levels derived from the three different curves described in the Mrak Report (Table 3).

If the figures for the three curves in Table 1 are utilized and the 1 in 1 million ($TD_{0.0001}$) as well as the 1 in 100 million ($TD_{0.000001}$) percent induction doses for DDT-caused tumors are considered according to the tabulated values for downward extrapolation, it can be seen from Table 3 that none of the three curves yields a dose, dispersed in the body, which reaches the concentration of 4×10^4 molecules per body cell. Even were we to assume that the approximate 1.5 kg daily food

TABLE 3

EXTRAPOLATIONS TO "SAFE DOSES" OF DDT WITH A TD_{50} OF 140 PPM IN THE DIET

Tumor Incidence on	Dose in Food (ppm)	Quantity in 1.5 Kg Food: Total Dose (µg)	Total Dose in Molecules	Molecules per Human Body Cell	Molecules per Human Liver Cell
Probit Curve					
$TD_{0.0001}$	0.18	270	4.6×10^{17}	3.3×10^{3}	9.2×10^{5}
$TD_{0.000001}$	0.056	84	1.4×10^{17}	1.0×10^{3}	2.8×10^{5}
Logistic Curve					
$TD_{0.0001}$	0.0014	2	3.4×10^{15}	24	6.8×10^{3}
$TD_{0.000001}$	0.000023	0.034	5.9×10^{13}	4.2×10^{-1} (or ~ 1 for 3)	1.2×10^{2}
One-Particle Curve					
$TD_{0.0001}$	0.0002	0.3	5.1×10^{14}	3.6	1.0×10^{3}
$TD_{0.000001}$	0.000002	0.003	5.1×10^{12}	3.6×10^{-2} (or ~ 1 for 27)	10

intake of a man would be uniformly contaminated with DDT and, in addition, that the whole "dose" of the compound were to enter the liver directly in unchanged form (both of which assumptions are obviously untenable), only extrapolations from the probit curve would result in values above those needed merely to be registered by the liver cells.

The Threshold of Certain Known Chemical Carcinogens.

Considerations related to those presented above were reported by the Food Protection Committee (1960), both in connection with Druckrey's 1959 feeding studies on rats using dimethylaminoazobenzene (DAB, or butter yellow) and with results from experiments on mice employing single injection doses of methylcholanthrene (MC), dibenzanthracene (DBA), and 3,4 benzpyrene (BP) (Bryan and Shimkin, 1943). Druckrey found that lifetime feeding of 0.3 mg of DAB daily to rats had no hepatic tumorigenic effect, whereas 1 mg of the dye fed daily over the life span of the animals resulted in a 20% incidence of hepatic tumors. In Bryan and Shimkin's single injection studies with mice, the "no-effect levels" for the production of sarcomas from the three compounds studied were established as follows: for MC, 4 ug; for DBA, 2 ug; and for BP, 30 ug.

The Committee pointed out that in Druckrey's studies a lifetime intake (summation of the daily dose for 700 days) of 10^{12} molecules DAB per rat liver cell was necessary to induce 20% incidence of hepatomas in the animals, whereas 10^{11} molecules

per cell was ineffective. To be more precise, however, it should be mentioned that the same total number of molecules produced some tumors when higher doses were administered for a shorter time period. Concerning the findings of Bryan and Shimkin with BP, they wrote: ". . . the injection of approximately 6 x 10^{16} molecules did not produce sarcomas, whereas the injection of 1.2 x 10^{17} molecules did" (Food Production Committee, 1960, p. 14). The Committee's comment on the difference between the molecular quantities of injected BP which had no tumorigenic effect and the quantities which did induce sarcomas is an important point.

Taking a slightly different approach to the DAB findings of Druckrey (1959), the individual daily dose where no effects were manifested may be considered; this dose is 0.3 mg/rat and it represents 5.7 x 10^{8} molecules per liver cell. The level at which 20% of Druckrey's animals developed hepatic tumors was 1 mg/rat/day--a presumed exposure of 1.9 x 10^{9} molecules per liver cell. According to the Food Protection Committee, one may assume that only 1% of the DAB reaches the liver unchanged, which would imply that the no-effect level is 5.7 x 10^{6} molecules per rat liver cell, whereas daily exposure of 1.9 x 10^{7} molecules per liver cell, over the life span of the rat, is necessary for induction of 20% incidence of hepatoma.

These figures regarding the minimal exposure to DAB through feeding which lead to liver tumors seem to be in excellent agreement with the data emerging from the extensive studies of

Terayama (1967) on DAB and its analogs. Following 50 mg singl intragastric administration to rats, Terayama found that with DAB itself--which has been described as "highly carcinogenic"--there was 28 ug of the compound bound to protein in a 7 g rat liver at the time of peak recovery. This would indicate that 5.4×10^7 molecules per liver cell were present. At the same dose level, the "weakly carcinogenic" analog of DAB-- 2'-methyl-4-monomethylaminobenzene (me-MAB)--gave a peak protein-bound dye recovery of only 11 ug per rat liver, while its carcinogenic activity was 1/6 that of DAB. Low tumor incidence thus resulted from 2×10^7 molecules of the compound per liver cell (cf. Druckrey, where 1.9×10^7 molecules per liver cell in the feeding experiments also led to a relatively low--20%--incidence of hepatoma). Finally, when the noncarcinogenic DAB analog--4-aminoazobenzene (AB)--was tested at the same dosage, protein-bound compound recovery in the rat liver was less than 2 ug. This quantity corresponds to 4.4×10^6 molecules of the chemical for each rat liver cell (cf. Druckrey's no-effect DAB dose of 5.7×10^6 molecules calculated earlier).

DAB and its analogs are the most thoroughly studie and probably the best understood of all known or suspected chemical carcinogens. The binding of the dye molecules to a highly specific protein in liver cell sap has been established. Speculations have been advanced that the dye-binding protein is a repressor or has the nature of an apo-repressor. In further experiments with DAB, Terayama found that as he lowered the starting dose, protein-

bound recovery was accordingly decreased, and the chemical ceased to act as a carcinogen. The lowest protein-bound recovery level for all three compounds mentioned in the foregoing (DAB, me-MAB, AB) was about 1 ug per rat liver.

Other Statistical Approaches.

Returning to the subject of proposals for statistical analysis of experimental data, on the basis of which downward extrapolations may be carried out to establish "safe" levels of human exposure to carcinogenic agents, some other methods should be discussed. In 1961, Mantel and Bryan utilized the findings of Bryan and Shimkin (1943) on MC in order to calculate a "virtually safe dose" (VSD) for methylcholanthrene. They defined VSD as an exposure level which would give rise to only 1 tumor in a population of 100 million individuals. They employed a probit curve with 99% confidence limit, arriving at a VSD for MC of 9×10^{8} mg (or 90 pg) per mouse. This same quantity was considered to be the VSD for man (Mantel and Bryan, 1961, p. 466). MC, unlike DAB, is not liver specific in its effects. If this dose were dispersed in the whole human body, the exposure would amount to 1 molecule for every 700 body cells. Should the 90 pg of the compound enter the liver in its totality, the VSD still would yield only 2 molecules for every 5 human liver cells.

In 1970, Schneiderman presented a somewhat different proposal for estimating an "acceptable risk dose" (ARD), which was incorporated into the Ad Hoc Committee's report as Appendix 3.

His procedure is based on use of the one-particle curve with an upper 95% confidence limit, and the lower section of the curve is constructed to pass through the origo (no dose, no response). From results obtained in animal experiments, he extrapolates only to the dose which should produce 1 tumor in 100,000 individuals ($TD_{0.001}$). On the other hand, when extrapolating from animal to man, he suggests: ". . . that an appropriate 'safety factor' for man should involve a reduction in the ARD for animals by a factor of 100 to allow for species' differences, another factor of 100 to allow for interactions with other carcinogens, and another factor of 100 to hedge against the incorrect choice of 'blow up' (weight or surface area) from animals to man. This would imply an ARD in man of about 1×10^{-6} the ARD in animals" (Schneiderman, 1970, p. 12).

Utilizing his assumptions, the ARD of DDT for man would be 3 pg total body dose, or 5.1×10^{9} molecules. This exposure, if dispersed throughout the body, would give 1 molecule for every 27,500 cells; or, if concentrated in the liver, 1 molecule for every 100 cells. It need hardly be emphasized that human exposure to such a quantity of this supposed carcinogenic pesticide does not appear to have any significance, especially in light of Dinman's work (Dinman, 1972) on threshold of effect for individual cells.

However, if one takes a converse approach and examines the exposures to DDT which would be "unsafe", accepting

Schneiderman's 10^{-6} safety factor for man and applying his preferred curve, a serious problem emerges. According to his method, the exposure which should cause tumors in 1 in 100 individuals (TD_1) is a total dose of 30 ng/man/day. In view of the fact that the whole human population of the earth has been ingesting at least 1000 times this quantity of the pesticide daily for the past 25 years, one would expect that were DDT indeed carcinogenic for man, and were these safety assumptions based in reality, there ought to have been a massive increase in primary liver cancer among humans in the age group at greatest risk; that is, those who are now between 50 and 70 years old. In fact, the average daily intake of DDT by man over these 25 years would correspond approximately to Schneiderman's TD_{50}; thus, in the population at risk, exposed for at least 1/4th of their lives, every 8th person should have developed hepatocarcinoma.

It is generally accepted that MC is a relatively potent carcinogen. When the socially acceptable risk is calculated according to Schneiderman's method, the ARD value for MC exposure is 3.15×10^{-6} pg. While the author admits that his procedure ". . . gives a somewhat more conservative answer than the procedure of Mantel and Bryan" (ibid, p. 11), this concession must be regarded as an understatement, considering that Mantel and Bryan's VSD for MC is 90 pg, a quantity which is 3×10^{7} fold greater than the ARD derived by utilizing Schneiderman's approach. The ARD value of 3.15×10^{-6} pg of MC, when expressed in terms of molecules, yields 1 molecule for every 20 billion cells in the

human body. Mathematical methods yielding such conclusions do not seem to have any relevance to human biology.

The newest version of these mathematical techniques was proposed by the FDA, published in the Federal Register (1973). Basically, the method involves a linear extrapolation downward from data in the observable range to a 1 in 100 million risk dose. However, for the lower ranges a more conservative slope is used, which results from application of the lower 90% confidence limit in constructing the curve. Starting once again from the findings of Bryan and Shimkin on MC (1943), the socially acceptable dose would be 120 ng, or 2 molecules of the compound for each body cell, if the probit line were to be extrapolated in a linear fashion to the $TD_{0.000001}$. However, since the slope of the lower portion of the curve is skewed to"make allowance for the sampling error", the acceptable exposure is reduced to 3.8 ng--that is, 1 molecule for every 16 body cells.

Concluding Remarks.

From the data discussed earlier, it seems probable that an hepatocellular threshold level for tumor induction exists at approximately 2×10^6 molecules per liver cell, irrespective of route of administration, at least for DAB and its analogs. If equally detailed information becomes available on other substances which have been demonstrated in well designed animal experiments to be strongly carcinogenic, it may become feasible to establish

a threshold exposure level for the carcinogenic transformation of cells.

We voice our agreement with the statement cited earlier from the Mrak Report (although for quite different reasons from those of the original writers) that in assessing safe levels of human exposure to chemical carcinogens ". . . it would be imprudent to place excessive reliance on mathematical sleight of hand" (Mrak Report, 1969, p. 495). Setting exposure standards on the basis of statistical extrapolations which fail to take account of fundamental biological concepts such as the principle of threshold can be highly misleading, resulting in unnecessarily conservative "permissible exposures", while at the same time failing to resolve the issue of determining the quantitative level at which chemical carcinogens will not have a deleterious effect on a living organism.

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MICHAEL M. BADEN, M.D.
520 FIRST AVENUE
NEW YORK, N.Y. 10016
TELEPHONE: 684-1600

2499

August 3, 1974

Mr. Arthur Smith
Vedder, Price, Kaufman, & Kammholz
39 South LaSalle Street
Chicago, Illinois 60603

Dear Mr. Smith:

I have obtained the causes of death in five additional Pottstown employees since the OSHA hearing of July 10, including the two outstanding in the vinyl chloride exposed group. I am therefore updating my previous report to you, having examined the causes of death of 351 of the 358 non-exposed employees and all of the 87 vinyl chloride exposed employees who have died since 1947 - during which time more than 750 workers were exposed to vinyl chloride.. One angiosarcoma of the liver was identified (in an exposed worker); there was no other cause of death in which vinyl chloride exposure could on the basis of the data be considered causative or contributory.

The very proper concerns expressed at the OSHA hearing as to "What is acceptable social and medical risk?" were with me the day following when I autopsied an eight-year-old boy who had drowned at a public beach, a 50-year-old window-washer who had fallen 35 stories and a number of victums of automobile accidents. Risks to health and life are a factor in all human activity and cannot be judged in vaccuo but rather are accepted in great measure depending upon degree of risk weighed against available alternatives - e.g.: close the beaches, prohibit outside window cleaning, ban the auto. We must certainly be constantly alert to identify any factors unwittingly introduced into the environment that increases such risks. However, the evidence against vinyl chloride in concentration of 50 ppm or less as a health risk to human beings is much less convincing than present evidence about cigarettes, alcohol, chloromycetin, smoked fish, electricity, sexual intercourse or even pregnancy - all of which we accept after considering alternatives. The structuring of categories of "informed-consent risk" ("volitional risk","having a choice") as contrasted to "uninformed-consent risk" ("non-volitional") is not to me as a physician as valid as might appear: does the cigarette smoker really understand (emotionally as well as intellectually) that he might get lung cancer? is an eight-year-old capable of understanding the risks of swimming? does the pedestrian struck by an auto have a choice? What is "free choice" if not a product of laws, mores, environment?

2351

Concern was very appropriately also expressed at the OSHA hearing as to potential unknown long-term effects of exposure to vinyl chloride. But this concern unfortunately can be made into an universal charge against all chemicals and medications and foodstuffs - Do we know the long-term consequences of food additives? of diet? of tetracycline? (indeed, the federal government even now sponsors programs to give methadone to pregnant women despite much concern among physicians that this drug may cause immediate and delayed adverse effects to the unborn child).

Reasonable people must make reasonable decisions on the basis of the best evidence at hand. Since 1947 one worker of 750 (87 of whom have died) exposed to vinyl chloride at levels much above the 50 ppm under present consideration developed angiosarcoma of the liver. Would the risks to life and health (mental and physical) be increased or decreased for vinyl chloride workers and their families if, to protect them, their jobs were to be eliminated (what were the poverty, malnutrition, psychiatric consequences of the well-intended but perhaps over-reactive governmental actions against mercury in swordfish or against cranberrys)? The social and economic factors necessary for proper physical and mental health must not be underestimated.

I think it reasonable to conclude, considering the evidence at hand and the alternatives available, that an acceptable threshold level of vinyl chloride exposure probably exists. And it is even possible, especially with present-day safety considerations including job rotation, that a 50 ppm exposure level provides appreciably less of a health risk than many other risks to health, industrial and otherwise, that our society has judged acceptable.

Sincerely,

Michael Baden

Michael M. Baden, M.D.

MMB:pw

A Consolidation of Chicago Wesley and
Passavant Memorial Hospitals

Wesley Pavilion

Superior Street and Fairbanks Court
Chicago, Illinois 60611

21, August 1974

R. S. Brookman, Manager
Research, Development and
Technical Service
Firestone Plastics Company
P.O. Box 699
Pottstown, Pennsylvania 19464

Dear Mr. Brookman:

We have revieved the data obtained by the analysis of approximately 300 employees in your Pottstown, Pennsylvania plant. In addition, we have conducted telephone interviews with representatives of the Quality Control Medical Laboratories, Inc. and the Metpath Laboratories.

Based on our experience in screening other industrial populations, and laboratory experience in general, we are prepared to offer the following opinions.

First, the major deviation from the normal pattern occurs in the gamma glutamyl transpeptidase (GGT) analysis. This enzyme, usually considered to be a sensitive test for many sorts of liver disease, is elevated in 22% of the workers. Upon closer inspection, one finds that the vast majority of abnormals have been generated by the Quality Control Laboratories, Inc. Telephone conversations with people from this lab have indicated that they use a "normal range" as published in the literature. It is our opinion that this is not desireable. Ordinarily one would develop their own "normal ranges" based on the analysis of a large number of people, preferably in a similar occupation, socio-economic range, and age and sex distribution. The Metpath Laboratories claim they developed their "normal ranges" by such a technique.

Therefore, we believe there is a reasonable question as to whether the "outlying values" from the Quality Control Laboratories, Inc. represent abnormalities in the patients or merely a minor misadjustment in the callibration of the test. The later premise is strengthened when one considered that there are very few abnormalities in other liver function tests, especially the alkaline phosphatase test, which usually has a high correlation with the GGT test.

A Member of The McGaw Medical Center of Northwestern University

It is our opinion that before any decisions on the presence or absence of liver disease in your employees is made, that you strongly consider repeating the GGT test in the people who were "abnormal". If possible, it would be desirable to have these employees analysed by both the Quality Control Laboratories and another independent laboratory.

The second part of our findings is that the other "abnormalities", i.e. protein electrophoresis, serum glutamic oxaalo acetic transaminase (SGOT), blood urea nitrogen (BUN), etc., are not increased above the level that one would expect in an industrial population of this age group.

Sincerely yours,

Kenneth A. Schneider, M.D.

Kenneth A. Schneider, M.D.
Director of Clinical Laboratories

KAS:mjg

CC: G. Kent, M.D.
L.H. Ballou, M.D.
J. Cassidy

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PETER M. NEMKOV
JOSEPH E. HADLEY
CAROLE C. HARRIS
WILLIAM W. PUGH

August 23, 1974

Mr. Julius Jimeno
Attn: OSH-36
Occupational Safety and
Health Administration
U.S. Department of Labor
Room 200
1726 M Street, N.W.
Washington, D. C. 20210

Re: In the Matter of: PROPOSED PERMANENT STANDARD FOR OCCUPATIONAL EXPOSURE TO VINYL CHLORIDE

Dear Mr. Jimeno:

On behalf of our client, The Society of the Plastics Industry, Inc. (SPI), we are herewith transmitting an original and four copies of a letter addressed to the undersigned from Joseph D. Banzer, Ph.D. of Diamond Shamrock Corporation.

The letter from Dr. Banzer is intended to be the response for Mr. Rodney P. Becker of Diamond Shamrock, a witness for SPI, to a request for information by the Solicitor during the Hearings (Tr. 677) and should be incorporated as a part of the official Record in the above-referenced matter.

If there is any question concerning this submission, please do not hesitate to contact us.

Respectfully submitted,

THE SOCIETY OF THE PLASTICS INDUSTRY, INC.

By Joseph E. Hadley
Joseph E. Hadley
Assistant General Counsel

Enclosures

nvironmental Labs | Diamond Shamrock Corporation
P.O. Box 348 Painesville, Ohio 44077/Telephone 216-352-9311

August 1, 1974

Mr. Joseph E. Hadley
c/o Keller & Heckman
1150 17th Street NW
Suite 1000
Washington, D.C. 20036

Dear Mr. Hadley:

Rod Becker, of our Plastics Division, has asked me to write to you concerning possible loss of vinyl chloride from charcoal during sample shipment. I have carried out an experiment in which a known amount of vinyl chloride was adsorbed on charcoal. The charcoal was transferred from the adsorption tube into glass bottles fitted with press-fit plastic caps and allowed to stand at room temperature. Duplicate samples were analyzed after 2, 5 and 14 days. No significant loss of vinyl chloride from the charcoal into the airspace above the charcoal was observed. The data are summarized in Table I.

Table I

Stability of Vinyl Chloride on Charcoal vs. Time

Time Interval, days	Vinyl Chloride Added, ppm	Vinyl Chloride Recovered
2 days	48 ppm	44 ppm (92%)
5	48	49 ppm (102%)
14	48	54 ppm (113%)

The data in Table I indicate there is no loss of vinyl chloride from charcoal with time. The relative range of error (-8% to +13%) is well within the ±20% required by OSHA for analyses at the 50 ppm level.

I must point out that the charcoal used in this experiment was specially prepared in our laboratory by grinding and reactivating commercial charcoal. Identical material is used both by us and our plants for all VCM analyses. However, I cannot be certain that all charcoals used by other analysts are as retentive as our material.

If you have any questions, please feel free to call me.

Sincerely,

Joseph D. Banzer

Joseph D. Banzer, Ph.D.
Senior Research Chemist
Environmental Testing
Diamond Shamrock Corporation

dp

cc: R. P. Becker - Technical Center

2697 138

Director, NIS
Through: Chief, HESB

April 26, 1974

Assistant Chief, HESB

Investigation of possible employee exposures to vinyl chloride monomer associated with the hot wire cutting of PVC meat and produce wrapping films.

1. In response to a request from your office to evaluate the possible emission of vinyl chloride monomer from hot wire cutting of PVC film, field measurements have been made.

2. In conjunction with a health hazard evaluation survey conducted in a suburban supermarket near Cleveland, Ohio an attempt to measure possible emission of vinyl chloride from a meat wrapping operation was made. A Wilks MIRAN infrared Gas Analyzer was used. Current/voltage fluctuations created by refrigeration equipment, etc. precluded the gathering of meaningful data.

3. The Kroger Company in Cincinnati, Ohio was subsequently contacted to arrange for vinyl chloride measurements in Cincinnati supermarkets. Mr. Phil Bouckhaert, Manager of Methods, Equipment, Supplies and Industrial Engineering, Kroger Company suggested that measurements be made at their 1014 Vine Street facility where they had several types of wrapping machines and PVC films.

4. On April 25, 1974 Mr. Jeffrey Lee and I conducted extensive vinyl chloride sampling at the Kroger facility utilizing their wrapping machines and film. Three films; Goodyear Choice Wrap: Borden BUF 61, and Borden TPH 87 were studied. Each film was studied over a wide range of cut-off-wire temperature under greatly exaggerated film cutting conditions. Fresh boxes of PVC film were also tested for vinyl chloride.

5. A Wilks MIRAN Infrared Gas Analyzer was utilized to make the measurements. Absorbance readings were made at wavelengths of 10.2 and 9.3 microns. Spectra between 8.0 and 14.5 microns were also examined.

6. The results of these field measurements were all negative. No vinyl chloride was found to be emitted by the hot wire cutting process or to be contained in film roll boxes. Under these operating conditions the lower limit of detection for the Wilks instrument was approximately 1 to 2 ppm of vinyl chloride.

7. A letter stating our findings and thanking the Kroger Company for their cooperation will be prepared and sent to Mr. Bouckhaert.

Robert VanKervort

MEMORANDUM

DEPARTMENT OF HEALTH, EDUCATION, AND WELFARE
PUBLIC HEALTH SERVICE
CENTER FOR DISEASE CONTROL
NATIONAL INSTITUTE FOR OCCUPATIONAL SAFETY AND HEALTH

: Mr. Alexander J. Reis
Associate Assistant Secretary for
National Programs, OSHA

DATE: March 26, 1974

: Jeffrey S. Lee
Special Assistant to Director
Division of Technical Services

LCT: Docket OSH-34
Exposure of Meatwrappers to Polyvinyl Chloride Film Decomposition Products

Your memorandum of February 19, 1974, to Dr. Key has been referred to me for action and reply.

The Division of Technical Services is continuing its investigation of the health problems of meatwrappers that are associated with the decomposition products of polyvinyl chloride. Specifically, we are cooperating in a study being conducted by the Center for Disease Control (CDC) and the Houston City Health Department. This study should define the prevalence of the associated astimatic-like syndrome and will provide a basis for future study design. Results from this initial investigation should be available by July.

We are also preparing a protocol for further, more extensive, environmental/medical investigations to be conducted under contract in FY '75, taking into consideration the findings of the Houston Study. These studies will also include more definitive evaluations of film decomposition products.

We are aware of several investigations that have been conducted to date on the volatile and semi-volatile thermal degradation products of PVC meat-wrapping film. I have attached these references for your information. The investigators have demonstrated that the plasticizer, dioctyl adipate/di-2-ethyl hexyl adipate and hydrogen chloride are the main contaminants from the hot wire cutting of PVC film. In addition, Boettner, et al., demonstrated that small amounts of over 50 other compounds, including vinyl chloride monomer, can be produced in the degradation of the pure PVC polymer. Within the near future, we will conduct an initial environmental evaluation to determine if vinyl chloride can be detected during meat wrapping operations. We will advise you as to the results of our investigations.

You mentioned that your "...field staff has been requested to conduct some studies on equipment and exposures." We would appreciate copies of their reports and would appreciate being advised of any future OSHA activity on this problem. We also welcome the assistance and cooperation of your staff in the design of future studies.

Dr. Phillip Polakoff, assisted by Mr. Robert Vandervort of our staff, have been assigned primary responsibility for the development and completion of the above-mentioned NIOSH efforts. In addition, Dr. Polakoff and Mr. Vandervort are preparing papers summarizing our current knowledge of the PVC heat wrapping problem. Please contact either of them directly at the above address or by phone (513-684-2732) if we can provide any further information or assistance.

Selected Bibliography

1. Bovee, H.H.; Monteith, L.E.; Orhein, R.M. (Department of Environmental Health, School of Public Health and Community Medicine, University of Washington, Seattle): "Thermal Decomposition of Meat Wrapping Film" Paper presented at the American Industrial Hygiene Conference, May 1971, Toronto, Canada.

2. VanHouten, R.W.; Cudworth, A.L.; Irvine, D.H. (Liberty Mutual Research Center, Hopkinton, Mass.): "Evaluation and Reduction of Air Contaminants Produced by Thermo Cutting and Sealing of PVC Packaging Film" Paper presented at the American Industrial Hygiene Conference, May, 1972, San Francisco, California.

3. Jaeger, R.J.; Hites, R.A.: Pyrolytic Evaporation of a Plasticizer from Polyvinyl Chloride Meat Wrapping Film. Bulletin of Environmental Contamination and Toxicology, Vol. 11, No. 1, 1974.

Additionally, the volatile combustion products of PVC resin and plastic have been carefully studied by:

4. Boettner, E.; Ball, G.; Weiss, B.: Analysis of the Volatile Combustion Products of Vinyl Plastics. Journal of Applied Polymer Science 13:377-391 (Feb. 1969).

THE PANTASOTE COMPANY OF NEW YORK, INC.
26 JEFFERSON STREET, PASSAIC, NEW JERSEY 07055
AREA CODE 201-777-8500 • TELETYPE 710-989-7108

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VIA SPECIAL DELIVERY,
REGISTERED MAIL -
RETURN RECEIPT REQUESTED

August 21, 1974

Ms. Joanne Goodell
Attention: Docket OSH-36
Occupational Safety & Health Administration
U. S. Department of Labor
1726 "M" Street N.W., Room 200
Washington, D. C. 20210

SUBJECT: Comments on the Department of Labor, OSHA Proposed Permanent Standard on Vinyl Chloride 29-CFR-1910.93q

Dear Ms. Goodell:

Following my letter of June 14, 1974 advising you of my intention to appear and present evidence at the OSHA Hearings on the above subject on behalf of The Pantasote Company, your office advised me on June 21 that written comments in lieu of appearance would be acceptable and would be a part of the record of the Hearings. The attached comments are submitted in accordance with that understanding.

As stated in these comments, the Proposed Standard would have a very serious effect on The Pantasote Company, and could, in fact, cause the shutdown of its plants. Since 90 percent or more of the activities of The Pantasote Company are dependent on the availability of polyvinyl chloride resin, the effect on the Company and its employees is self-evident. In view of the vital concern we have for the repercussions of the promulgation of the Proposed Standard, we trust that the Secretary will give serious and careful consideration to our comments.

Sincerely yours,

THE PANTASOTE COMPANY

Jacob Jaglom
Group Vice President

JJ/nm
Attachment

2702

THE PANTASOTE COMPANY

Comments on the Department of Labor, OSHA

Proposed Permanent Standard on Vinyl Chloride

29-CFR-1910.93q

August 21, 1974

INTRODUCTION:

The Pantasote Company, through its wholly owned subsidiary, The Pantasote Company of New York, Inc., operates two polyvinyl chloride resin manufacturing plants in Passaic, New Jersey and Point Pleasant, West Virginia, employing approximately 150 persons, and producing a total of approximately 160 million pounds of polyvinyl chloride resin. The Pantasote Company of New York owns the Passaic, New Jersey plant, and shares the ownership of the West Virginia plant equally with The General Tire & Rubber Company of Akron, Ohio. The Pantasote Company utilizes its share of the production of the two plants almost totally within the Company, using the polyvinyl chloride resin to produce PVC compounds, film and sheeting, refrigerator gaskets, automotive molded parts, baby pants, packaging trays, and printed and laminated constructions for wallcovering, shower curtains, and many other applications. About 90 percent of the 3,000 employees of The Pantasote Company depend for their livelihood on the availability of polyvinyl chloride resin produced by its Pantasote Company of New York subsidiary. Approximately 85 percent of the sales of The Pantasote Company, which in 1973 were $86 million and in 1974 will exceed $100 million, consist of products derived from the polyvinyl chloride resin.

The provisions of the Proposed Standard affect The Pantasote Company as:

a. A resin manufacturer.

b. A manufacturer of pelletized compounds.

c. A resin processor by means of calendering, extrusion, and injection molding.

RESIN MANUFACTURE

The Pantasote Company recognizes that available evidence directs strong suspicion at vinyl chloride as an occupational hazard. We do not agree, however, that medical evidence justifies a Standard requiring no detectable levels of vinyl chloride monomer in the work areas. Nor can we find anywhere the evidence that would indicate that such a Standard is either technically or economically feasible. We agree that prudence dictates a reduction of vinyl chloride monomer exposure to the lowest feasible levels. The Pantasote Company, therefore, supports the first stage of the proposal of The Society of the Plastics Industry to reduce exposure levels to 40 ppm ceiling and 25 ppm maximum daily time weighted average. We support this position with the reservation that we do not clearly know the feasibility or the cost of attaining these exposure goals. We are firmly convinced that our plants--closed plants in northern locations--cannot comply with the "no detectable level" requirement and would have to be shut down. The attainment of the lower exposure levels of the SPI proposals may require major modifications of the plants at a cost that may be beyond the resources of this Company.

The suspension process utilized by Pantasote, which is typical of a large portion of the industry, is a batch process requiring the opening of reactors and reactor entry, and having many points for potential emission of VCM in the dozens of valves, pumps, flanges, manheads, and transfer points.

The monitoring in our plants to date indicates that it is essential that a time-weighted average approach be utilized in order to allow for

the orderly operation of the plants. Many causes bring about frequent momentary excursions above the average and a ceiling approach at a level below 40 ppm would necessitate the use of respirators on a continuous basis, a totally impractical method of plant operation, as testified to by many expert witnesses.

We do not want to burden the already lengthy record with further comments on the detailed provisions of the Standard. Numerous comments have been made on the need to refine the requirements for monitoring, the relaxation of provisions for respiratory protection to include canister-type respirators, and the vital need to revise the labeling requirements from the language which will cause fear and panic among workers and users of vinyl chloride and polyvinyl chloride, to constructive directions for proper action in case of emergency.

Much has been said about the impracticality of impervious suits, about the difficulty to operate in cumbersome clothing and equipment inside the vessels and during maintenance and repair operations. Such operations can be carried out safely with ordinary work clothing and with canister-type respirators. The record-keeping requirements are burdensome and unproductive. An occupational hazard which is thought to have a latency period of 15 to 20 years does not warrant the requirements of medical attention or the reporting to OSHA of every incident of vinyl chloride emission within 24 hours of occurrence.

PELLETIZED COMPOUNDS

As a major manufacturer of pelletized compounds, Pantasote is vitally concerned with the effect of the Proposed Standard on its

customers. Determinations of the VCM content of pelletized compounds show that the pelletizing process reduces residual VCM to levels ranging from non-detectable in plasticized PVC compounds to less than 100 ppm in unplasticized PVC compounds. The monitoring of processing plants utilizing pelletized compounds showed no detectable levels of VCM. Pantasote urges, therefore, that pelletized compounds be included within the definition of "Fabricated Products," and that the Standard not apply to processors of pelletized compounds.

FABRICATING OPERATIONS

Our data indicate that exposures to vinyl chloride in our fabricating operations (calendering, extrusion, and injection molding) are at extremely low levels. The Hearing Record contains additional voluminous testimony to this point. In the circumstances the burdensome provisions of the Standard should not, and need not, apply to fabricating operations such as ours. We might add that the ability of OSHA to monitor compliance with the Standard would be tremendously diluted if it were to extend the provisions of this Standard to the very large number of locations of the industry comprising fabricating operations. We strongly recommend that the provisions of any Permanent Standard apply only to monomer and polymer manufacturing operations.

ECONOMIC CONSIDERATIONS

It is vitally important that the Department of Labor recognize that the VCM and PVC resin industry is composed principally of very large corporations with considerable resources. Pantasote, along with only two or three other companies, is a relatively small company with

limited resources, competing with companies many, many times its size. The economic burdens imposed by this Standard would weigh heavily on a Company of our size, could place a great strain on our resources, and impair our ability to compete. Our ability to survive will be greatly affected by the length of time granted us to comply with the provisions of the Standard and by the availability of financing for the necessary monitoring equipment and engineering modifications of our plants. The Department of Labor must give special consideration to the circumstances of the smaller companies, or their very survival could be involved.

CONCLUSIONS

The Pantasote Company recognizes its responsibility to its employees and its customers to provide safe working conditions and to produce safe products. However, the Standard that is promulgated must be justified by the evidence and must be technically and economically feasible. The Proposed Permanent Standard meets none of these requirements. It will bring about the shutdown of the vital PVC industry unless it is radically revised to bring it within the realm of feasibility.

The fact that the incidence of angiosarcoma is confined to four of the five oldest plants in the United States, and principally to two of these plants, is strong evidence that unusual operating conditions in these plants, rather than the hazard of vinyl chloride monomer at low levels of exposure, were the principal cause of angiosarcoma. Many plants have operated for many years with no incidence of angiosarcoma. A large body of evidence was adduced during the Hearings demonstrating that vinyl chloride is not unsafe at concentrations of 50 ppm time weighted average.

The cases of angiosarcoma being ascribed to fabricating operations are so poorly documented as to be of no evidentiary value whatever. Were the sensitivity of humans to vinyl chloride exposure as great as such cases would suggest, angiosarcoma would be a disease of truly epidemic proportions rather than the rare disease affecting a small number of persons over a great many years.

Voluminous evidence supports the conclusion that processors of PVC resin and compounds should be excluded from the provisions of the Proposed Standard in the light of the extremely low levels of VCM found in these plants.

Special consideration must be given to the smaller PVC producers, such as Pantasote, to enable them to comply with the requirements of the Standard within a time period commensurate with their resources, or by assisting with the necessary financing.

Submitted By:

Jacob Jaglom, Group Vice President

The Pantasote Company
26 Jefferson Street
Passaic, New Jersey 07055

Date: August 21, 1974

Lassiter

2763

146

Industrial BIO-TEST Laboratories, Inc.
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DECATUR, ILLINOIS 62526

TOXICOLOGY
ENVIRONMENTAL SCIENCES
CHEMISTRY
PLANT SCIENCES
MEDICAL SCIENCES

AREA CODE 217
TELEPHONE 877-9254

August 16, 1974

Dr. Kenneth D. Johnson
Manufacturing Chemists Association
1825 Connecticut Avenue
N. W. Washington, D. C. 20009

Dear Dr. Johnson:

Enclosed are tables summarizing mortality, body weight and gross pathologic findings through the eleventh month of inhalation exposures on the chronic inhalation toxicity study with Vinyl Chloride. I've also included a table giving daily concentration data from the seven-month point to the end of the eleventh month.

Preceding each table is a brief discussion of the data. A complete and formal eleven-month status summary with full evaluation of this data plus histopathologic data will be submitted shortly.

Sincerely,

Ken Schadeberg

Ken Schadeberg, B.S.
Senior Group Leader
Inhalation Toxicity

KS/dm
enc.

cc - Dr. T. R. Torkelson
J. W. Goode
M. L. Keplinger

1. Analytical Concentration Data

The daily range and mean of the Vinyl Chloride concentrations in each chamber from exposure 153 (data previous to this exposure given in seven-month status report) through the end of the eleventh testing month are reported in Table I. The overall average daily vapor concentrations were calculated to be 49.9, 198, 2501 and 2489 ppm for the T-I through T-IV chambers, respectively.

TABLE I

TEST MATERIAL: Vinyl Chloride (Ethylene Derived)

Chronic Vapor Inhalation Toxicity Study - Albino Rats, Albino Mice and Golden Hamsters

Chamber Concentration Data

Exposure Number	Chamber Concentration (ppm) T-I Range		T-I Mean	T-II Range		T-II Mean	T-III Range		T-III Mean	T-IV Range		T-IV Mean
153	45.5	48.5	47.5	176	214	197	1820	2680	2467	2340	2500	2409
154	50.5	55.0	51.5	180	242	206	2300	2820	2459	2400	2640	2493
155	Holiday, no exposures											
156	43.5	53.5	46.5	180	242	202	2180	3000	2554	2140	2380	2231
157	46.5	52.5	50.4	154	194	184	2320	2600	2457	2160	2400	2294
158	36.5	52.5	45.0	138	250	192	1440	2600	2240	2500	3020	2760
159	44.5	55.0	49.2	130	138	134	2460	2620	2540	2600		2600
160	38.0	67.5	45.1	124	260	196	2000	3120	2687	1100	2510	2264
161	47.0	61.0	53.1	130	200	179	2380	2920	2384	2500	3000	2664
162	47.5	53.0	57.8	150	232	181	2380	2700	2484	2340	2700	2456
163	50.0	53.5	51.5	180	210	195	2440	2800	2580	2740	3040	2815
164	47.0	53.0	52.1	184	202	187	2200	2660	2488	2600	2900	2669
165	46.5	56.0	49.4	190	216	200	2420	2800	2583	2660	2800	2755
166	47.0	71.0	52.3	184	228	201	2100	2820	2520	2160	2500	2353
167	42.5	53.0	47.9	180	204	199	2300	2740	2479	2480	2940	2667
168	38.0	55.5	49.4	180	226	193	2160	2800	2472	2200*	2800	2545
169	44.0	51.0	47.6	196	215	207	2440	3710	2932	2500	2900	2729
170	48.0	56.5	53.6	184	216	205	2120	3100	2528	2460	2980	2668
171	46.0	51.5	49.3	186	198	191	2260	2620	2487	2400	2580	2488
172	47.0	50.5	48.5	202	214	209	2180	2600	2478	2140	2460	2339

TABLE I continued

TEST MATERIAL: Vinyl Chloride (Ethylene Derived)

Chronic Vapor Inhalation Toxicity Study - Albino Rats, Albino Mice and Golden Hamsters

Chamber Concentration Data

ay iber	Exposure Number	Chamber Concentration (ppm) T-I Range		T-I Mean	T-II Range		T-II Mean	T-III Range		T-III Mean	T-IV Range		T-IV Mean
	173	44.5	55.5	50.4	154	220	197	1500	3340	2468	2340	2760	2532
	174	46.5	53.0	47.9	180	222	194	2100	2560	2367	2480	2600	2512
	175	48.5	52.5	50.5	194	206	199	2380	2780	2487	2460	2520	2496
	176	45.0	54.5	48.8	182	210	198	2320	2760	2534	2320	2660	2536
	177*	47.5	55.0	51.0	-	-	-	2440	2780	2618	2560	2820	2673
	178	47.5	52.5	51.2	186	222	203	2420	2700	2573**	2580	2700	2640
	179	44.0	56.0	49.4	164	236	201	2560	2740	2645	-	-	-
	180	Equipment malfunction, no exposures											
	181***	45.5	57.0	51.4	188	218	204	1900	2760	2347	1900	2760	2347
	182	44.0	51.5	50.0	184	212	193	2320	2500	2396	2420	2500	2464
	183	43.0	75.0	57.4	88	222	171	2000	2640	2364	2100	2620	2280
	184	47.0	55.0	50.9	172	230	203	2300	2720	2501	2040	2460	2355
	185	39.0	57.5	48.0	180	214	199	2140	2400	2328	2280	2500	2330
	186	35.0	66.0	48.6	182	250	209	2140	2888	2397	2200	2600	2392
	187	38.0	75.5	49.9	170	266	202	2140	3240	2537	2500	2560	2545**
	188	43.0	50.0	47.4	196	224	205	2160	2560	2400	2460	2560	2524
	189	Holiday, no exposures											
	190	41.0	50.5	47.5	192	210	198	2300	2620	2431	-	-	-
	191	45.5	58.5	50.2	192	251	196	2160	2540	2350	2240	2320	2272
	192	46.0	57.0	50.5	182	214	200	2320	2620	2452	2380	2440	2400
	193	41.0	55.5	50.5	18[illegible]	234	215	2380	2880	2633	2420	2760	2540

TABLE I continued

TEST MATERIAL: Vinyl Chloride (Ethylene Derived)

Chronic Vapor Inhalation Toxicity Study - Albino Rats, Albino Mice and Golden Hamsters

Chamber Concentration Data

Day Number	Exposure Number	Chamber Concentration (ppm) T-I Range		T-I Mean	T-II Range		T-II Mean	T-III Range		T-III Mean	T-IV Range		T-IV Mean
270	194	48.0	54.5	50.1	172	214	204	2260	2660	2447	2140	2400	2270
271	195	43.5	50.5	47.3	190	208	200	2140	2560	2439	2380	2500	2408
274	196	44.5	51.0	47.9	188	216	202	2180	2580	2377	2400	2460	2440
275	197	38.0	56.0	49.5	174	242	198	2160	2640	2386	2440	2440	2440
276	198	45.5	52.5	48.9	188	214	199	2320	2720	2457	2340	2740	2504
277	199	47.0	56.0	50.3	170	220	201	2280	2860	2585	2300	2920	2656
278	200	37.0	52.5	48.3	176	226	200	2220	2700	2473	2580	2900	2672
281	201	40.0	56.0	47.5	168	232	204	2360	2740	2515	2520	2720	2600
282	202	43.5	54.0	48.2	176	236	202	2120	2940	2459	2280	2520	2413
283	203	43.0	54.0	49.7	188	216	206	2340	2920	2550	2540	2780	2640
284	204	47.0	57.0	52.5	144	220	194	1400	3420	2620	2210	2600	2478
285	205	43.0	58.5	50.3	154	224	199	2100	2980	2504	2140	2560	2397
288	206	Equipment malfunction, but animals were exposed											
289	207	42.5	59.0	48.5	194	206	200	2360	2820	2593	2480	2620	2528
290	208	40.0	53.5	45.9	204	218	209	2280	2820	2482	2280	2560	2424
291	209	45.5	51.5	49.3	166	220	203	2380	2680	2521	2440	2560	2516
292	210	Equipment malfunction, no exposure											
295	211	46.5	54.5	47.6	186	230	207	2280	3200	2577	2280	2520	2428
296	212	43.5	53.0	49.3	200	210	205	2480	2660	2613	2520	2680	2585
297	213	Vinyl Chloride gas regulator broken, no exposures											

TABLE I continued

TEST MATERIAL: Vinyl Chloride (Ethylene Derived)

Chronic Vapor Inhalation Toxicity Study - Albino Rats, Albino Mice and Golden Hamsters

Chamber Concentration Data

Day Number	Exposure Number	Chamber Concentration (ppm) T-I Range	T-I Mean	T-II Range	T-II Mean	T-III Range	T-III Mean	T-IV Range	T-IV Mean
28	214	Holiday, no exposures							
29	215	Holiday, no exposures							
32	216	Vinyl Chloride gas regulator broken, no exposures							
33	217	Vinyl Chloride gas regulator broken, no exposures							
34	218	45.5 50.5	48.2	202	226	218	2140 2460	2288	2400 2500	2480
35	219	44.5 52.5	48.6	186	210	204	2280 2740	2565	2400 2520	2445
36	220	45.0 57.5	42.3	190	218	203	2360 2660	2481	2400 2500	2440
39	221	41.5 49.5	45.9	168	192	185	2080 2500	2258	2360 2500	2410
40	222	47.5 55.0	51.0	186	196	193	2280 2500	2380	2280 2500	2364
41	223	44.5 57.5	51.3	160	226	184	1 sample only	2280	1 sample only	2440
42	224	39.0 46.0	42.8	148	172	160	1800 2040	1920	2640 2960	2800
45	225	37.0 52.5	44.2	146	196	174	1800 2440	2073	2500 3100	2680
46	226	46.0 55.0	54.0	182	220	208	2080 2500	2473	2400 2620	2480
47	227*	46.5 55.5	50.6	152	242	205	2040 2800	2558	- -	-
48	228	46.5 52.0	49.2	170	280	192	1960 2380	2022	2240 2580	2400
49	229	44.5 52.0	49.1	192	222	201	2440 2620	2542	2500 2720	2640
52	230	49.5 56.5	52.0	198	222	210	2500 2680	2699	2300 2620	2460
53	231	49.5 62.0	54.9	176	250	208	2160 2840	2475	2260 2380	2333
54	232	49.5 54.5	50.7	200	208	204	2220 2620	2405	2200 2460	2348
55	233	45.5 56.5	49.7	190	206	196	2400 2840	2597	2220 2500	2370

Industrial BIO-TEST Laboratories, Inc.

TABLE I continued

TEST MATERIAL: Vinyl Chloride (Ethylene Derived)

Chronic Vapor Inhalation Toxicity Study - Albino Rats, Albino Mice and Golden Hamsters

Chamber Concentration Data

ay ber	Exposure Number	Chamber Concentration (ppm) T-I Range		T-I Mean	T-II Range		T-II Mean	T-III Range		T-III Mean	T-IV Range		T-IV Mean
	234	48.0	62.0	54.0	170	238	202	2300	2760	2576	2520	2760	2570
	235	Equipment malfunction, but animals exposed											
	236*	-	-	58.0	200	204	202	2500	2580	2540	-	-	-
	237*	53.5	58.5	55.5	186	198	191	2220	2500	2376	-	-	-
	238	56.0	56.0	56.0	206	208	208	2260	2380	2300	1 sample only		2420
	239	47.5	55.0	51.3	185	210	202	2160	2600	2556	1700	2300	2084

No exposure in group indicated; animal cages repaired.
Exposures cut to 5½ hours; animal cages repaired.
Exposures cut to 4½ hours; gas chromatograph repaired.
: In exposures noted above, the equipment malfunction is referring to a breakdown in the gas chromatograph or ential sampler valve system-chamber concentrations were believed to be near targets.

II. Mortality Data

The cumulative monthly mortality data are presented in Table II. Below is a brief discussion of the data for each species.

Rats: With the exception of the T-IV rats (composed of females only and permitted food ad libitum) there is a dose related mortality effect in both sexes at the eleven month point. Although the total number of rats which died in the T-IV group is not as great as in the T-III female group, there was a rapid increase in mortality during the eleventh month in the T-IV group.

Mice: Dose related mortality effects were observed in both male and female mice at the eleven month point.

Hamsters: A significant increase in mortality rate was observed among male and female T-III hamsters during the eleventh month of the investigation. There were no other significant mortality findings.

TABLE II

TEST MATERIAL: Vinyl Chloride (Ethylene Derived)

Chronic Vapor Inhalation Toxicity Study - Albino Rats, Albino Mice and Golden Hamsters

Summary of Mortality Data - Albino Rats

Month Number	Accumulative Number of Mortalities at End of Month								
	Control		T-I		T-II		T-III		T-IV
	M	F	M	F	M	F	M	F	F
1	0	0	0	0	0	0	0	0	0
2	0	0	2	0	1	0	0	0	0
3	0	1	2	0	1	0	1	4	0
4	0	2	4	1	3	0	1	4	0
5	2	3	5	1	6	1	3	4	0
6	3	4	6	2	6	1	5	4	0
7	4	4	8	6	6	2	6	5	0
8	4	5	9	7	10	5	10	8	1
9	4	6	10	8	11	5	16	15	4
10	5	7	11	11	12	9	24	28	7
11	7	8	13	13	14	19	34	52	17

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TABLE II continued

TEST MATERIAL: Vinyl Chloride (Ethylene Derived)

Chronic Vapor Inhalation Toxicity Study - Albino Rats, Albino Mice and Golden Hamsters

Summary of Mortality Data - Albino Mice

Month Number	Accumulative Number of Mortalities at End of Month							
	Control		T-I		T-II		T-III	
	M	F	M	F	M	F	M	F
1	0	0	0	0	0	0	0	0
2	0	0	0	3	0	0	0	0
3	2	0	6	4	3	0	1	0
4	5	1	19	5	4	1	4	7
5	8	2	26	6	4	2	8	7
6	12	2	30	6	8	3	10	9
7	18	5	31	11	9	12	13	23
8	18	5	32	14	16	30	30	46
9	23	8	38	24	37	58	56	75
10	28	10	42	31	78	82	76	98
11	30	15	54	46	96	94	100*	100*

* All surviving mice sacrificed

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TABLE II continued

TEST MATERIAL: Vinyl Chloride (Ethylene Derived)

Chronic Vapor Inhalation Toxicity Study - Albino Rats, Albino Mice and Golden Hamsters

Summary of Mortality Data - Golden Hamsters

Month Number	Accumulative Number of Mortalities at End of Month							
	Control		T-I		T-II		T-III	
	M	F	M	F	M	F	M	F
1	5	4	4	4	7	5	2	5
2	8	11	10	12	9	16	10	11
3	8	13	12	14	12	18	14	13
4	9	15	16	16	15	19	15	13
5	9	18	16	16	16	20	15	15
6	13	18	16	16	20	21	16	16
7	13	28	24	19	22	33	18	26
8	14	28	24	19	23	35	19	28
9	14	29	24	20	25	36	20	28
10	14	30	26	20	26	37	23	29
11	15	31	28	21	26	38	28	36

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III. Body Weight Data

The mean body weight and weight gain data are presented in Tables III through V. Below is a brief discussion of the data for each species.

Rats: There are no dose related findings.

Mice: There appeared to be a drop in body weights of both the males and females of the T-III group during the tenth month of testing. These mice were sacrificed during the eleventh testing month. The other test levels do not indicate any definite adverse effecs.

Hamsters: Except for th T-III female hamsters, which revealed a low mean body weight for the eleventh month, there are no significant findings.

Proliferation of sinusoidal lining cells. Thrombosis

TABLE III

TEST MATERIAL: Vinyl Chloride (Ethylene Derived)

Chronic Vapor Inhalation Toxicity Study - Albino Rats, Albino Mice and Golden Hamsters

Summary of Mean Body Weights - Albino Rats

	Mean Body Weights (grams) Month Number:												
Group	0	1	2	3	4	5	6	7	8	9*	10	11	Gain
Control	205	363	456	498	517	537	543	550	559	697	677	689	484
T-I	209	346	448	491	526	545	554	567	593	651	640	670	461
T-II	201	360	457	490	522	549	567	565	585	681	663	696	495
T-III	200	351	453	499	529	550	567	576	581	627	629	680	480
Control	160	235	275	299	315	320	326	344	356	392	366	380	220
T-I	168	232	274	297	310	308	323	348	367	357	363	374	206
T-II	171	242	289	302	313	316	329	342	358	368	362	386	215
T-III	165	240	287	303	312	321	329	346	359	359	356	374	209
T-IV	181	251	282	288	291	314	324	338	348	363	363	384	203

*Apparent malfunction of animal weighing machine during month indicated.

TABLE IV

TEST MATERIAL: Vinyl Chloride (Ethylene Derived)

Chronic Vapor Inhalation Toxicity Study - Albino Rats, Albino Mice and Golden Hamsters

Summary of Mean Body Weights - Albino Mice

	Mean Body Weights (grams) Month Number:												
Group	0	1	2	3	4	5	6	7	8	9*	10	11	Gain
Control	35	40	41	43	46	43	46	52	49	47	43	47	12
T-I	33	39	39	43	44	42	47	45	47	47	38	48	15
T-II	36	40	38	42	44	42	45	43	44	46	37	44	8
T-III	36	38	40	43	44	38	45	41	41	43	31	**	**
Control	29	32	33	35	36	35	38	37	41	40	37	40	11
T-I	30	34	34	37	39	38	40	41	40	40	42	43	13
T-II	28	31	34	35	37	36	38	36	40	39	30	40	12
T-III	28	31	34	35	36	37	37	34	37	39	27	**	**

*Exposures were terminated for all mice at the end of the ninth month of the investigation.
**All surviving T-III mice were sacrificed during the eleventh month of the investigation.

TABLE V

TEST MATERIAL: Vinyl Chloride (Ethylene Derived)

Chronic Vapor Inhalation Toxicity Study - Albino Rats, Albino Mice and Golden Hamsters

Summary of Mean Body Weights - Golden Hamsters

		Mean Body Weights (grams) Month Number:												
ex	Group	0	1	2	3	4	5	6	7	8	9	10	11	Gain
ale	Control	73	98	110	120	129	130	133	130	138	141	129	126	53
	T-I	71	90	101	114	120	121	122	128	126	118	115	110	39
	T-II	63	89	94	99	119	108	120	118	122	117	115	115	52
	T-III	60	[illegible]	91	93	91	88	112	110	115	115	115	114	54
emale	Control	77	104	108	118	120	121	124	125	128	133	119	116	39
	T-I	78	95	102	112	118	118	122	130	126	125	118	110	32
	T-II	74	94	99	108	107	106	114	112	116	118	112	112	38
	T-III	69	92	100	106	106	104	110	118	105	107	103	99	30

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IV. Gross Pathology

A summary of tumor data based on gross pathologic findings among all species is presented in Table VI. This table does not take into account animals that died and postmortem autolysis or cannibalism precluded gross pathologic examination.

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TABLE VI

TEST MATERIAL: Vinyl Chloride (Ethylene Derived)

Chronic Vapor Inhalation Toxicity Study - Albino Rats, Albino Mice and Golden Hamsters

Summary of Tumor Data
Based on Gross Pathologic Findings
Among All Species Through 8/14/74

Species	Group	Number of Animals Examined			Tissue Involved (Male/Female)			
		Total	Male	Female	Liver	Lung	Skin*	Kidney
Mice	Control	4	3	1	0/0	1/1	0/0	0/0
	T-I	36	12	24	4/9	5/16	2/7	0/1
	T-II	133	67	66	45/57	52/39	7/26	5/9
	T-III	121	58	63	48/47	44/45	4/20	7/3
Rats	Control	0	0	0	0/0	0/0	0/0	0/0
	T-I	3	3	0	2/0	2/0	0/0	0/0
	T-II	11	4	7	0/5	0/4	0/1	0/0
	T-III	52	18	34	7/24	0/6	0/8	0/2
	T-IV	7	0	7	0/5	0/0	0/5	0/0
Hamsters	Control	0	0	0	0/0	0/0	0/0	0/0
	T-I	0	0	0	0/0	0/0	0/0	0/0
	T-II	0	0	0	0/0	0/0	0/0	0/0
	T-III	10	7	3	2/2	4/1	1/0	0/0

* Mammary Tumors

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ALTERATIONS OF LIVER AND SPLEEN AMONG WORKERS EXPOSED TO VINYL CHLORIDE

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The observation that an environmental factor causes a lesion raises many problems, such as the identification of the lesion, the establishment of a causal connection, the recognition of the incidence, the discovery of the precursor lesion, the understanding of the pathogenesis of the process, and last but not least, information about general pathophysiologic processes for which the environmentally induced process represents a useful model. The recent discovery[1] that angiosarcoma of the liver, a tumor otherwise very rare, develops in relatively high frequency in workers exposed to gaseous vinyl chloride in its polymerization process to the widely used polymer raised the above mentioned problems. The occurrence of angiosarcomas in such workers was called to our attention by the virtually simultaneous submission of pertinent pathological material to the pathological laboratories of the National Cancer Institute by Drs. H. Falk and M.M. Key and of the Mount Sinai School of Medicine by Dr. I. Selikoff only three months ago. An investigation of the problem, mainly morphologic, was initiated and the preliminary results form the basis of this report which concerns mainly the precursor stage of a peculiar hepatic fibrosis but which is supplemented by both speculations and by coordination with information in the literature. Gratitude is expressed to the mentioned providers of the material, who subsequently also included Dr. C.W. Heath, Jr., all of whom are presenting their own observations at this conference. Thanks are also due to the many pathologists who observed the

material originally and permitted its utilization in this preliminary survey. It is by necessity incomplete because the clinical data have not been fully evaluated by us, although many are presented at this conference. Moreover, the pathological analysis was restricted to the study of the liver and spleen specimens available to us. In a companion study one of us (LBT) will present subsequently the features of angiosarcoma on the same material as a continuation of this report.

Our recognition and investigation of the precursor lesion was stimulated by a German study[2] coming to our attention at the same time in which a peculiar type of hepatic fibrosis was recognized in vinyl chloride - polyvinyl chloride workers investigated for acro-osteolysis.[3] Their study was based on histologic and laparoscopic observations and is also presented at this meeting. It thus shares with the initial recognition of the relation between vinyl chloride exposure and hepatic angiosarcoma[1] the credit of motivating us to delineate the precursor stage. This was further augmented by the opportunity of a visit to the German investigators in Bonn early in this investigation.

MATERIAL AND METHODS

The following report is based on the histologic study of autopsy and biopsy material of the liver from 11 patients who were exposed to vinyl chloride (VC) in the polymerization of polyvinyl chloride (PVC) (Table 1). Five of these patients did not have an angiosarcoma of the liver. Most of their liver biopsies were obtained during laparotomy performed because of manifestations of portal hypertension. One of these 5 patients had a second liver biopsy approximately 2 years after the initial biopsy which was taken at the time of splenectomy and is the subject of the report by Dr. P. Berk. In addition, suitable histological material was available for the study of the hepatic fibrosis and other precursor lesions from 6 patients who had hepatic angiosarcomas. In two of them, liver biopsies had been performed before the angiosarcoma was diagnosed and in several others both biopsy and autopsy materials were submitted so that a total of 12 specimens were available for detailed histologic observations from these 6 patients (Table 1).

The incidence of each lesion was recorded depending on the opportunity to ascertain it. Hematoxylin and eosin stains, connective tissue stains such as Masson trichrome and chromatrope aniline blue, and silver impregnations were employed as well as PAS reaction after removal of glycogen by diastase. In five of the 11 patients, 2 with angiosarcoma and 3 without angiosarcoma, splenic tissue was available for examination and the same stains were performed.

The material submitted also included two cases of primary hepatic carcinoma. One was an autopsy specimen of an eight-year-old girl, the daughter of one of the workers in the factory engaged in the polymerization of VC. The other was biopsy material of a 54-year-old worker who for 17 years laminated PVC sheets.

Through the courtesy of Drs. P.J. Scheuer and M. Schmid it was possible to examine for comparison also three surgical specimens of liver of patients with portal hypertension following prolonged exposure to Fowler solution in the treatment of psoriasis. Two of these cases have been reported in detail.[4]

OBSERVATIONS

Livers Without Angiosarcoma

Fibrosis. The portal tracts were enlarged to varying degree, some appearing almost normal and others being conspicuously widened by the presence of excess connective tissue. This usually resulted in a round shape on the cut surface (Fig. 1). Irregular connective tissue septa radiated only exceptionally into the parenchyma. These septa usually linked portal tracts with each other, but occasionally formed connections between central and portal canals. The lobular architecture was thus entirely preserved and the central canals were devoid of excess fibers. In three of the specimens, septa formation was entirely absent. On higher magnification the portal tracts

varied in their appearance in the same specimen. Where excess connective tissue was found, it frequently separated liver cell plates in the periportal zone to result in a disruption of the limiting plate of the parenchyma (Fig. 2). In many instances the excess connective tissue consisting of dense collagenous bundles contained relatively few cells. Frequently it was arranged around proliferated bile ductules with abnormal epithelium and sometimes this periductular fibrosis was so intense as to resemble the picture of a congenital hepatic fibrosis[5] (Fig. 3). In other places a moderately dense lymphocytic infiltration intermixed with some histiocytes was noted (Fig. 4). The branches of the portal vein were often surrounded by dense connective tissue which sometimes infiltrated the wall of the larger veins separating the smooth muscle and elastic fibers (Fig. 4). The endothelial lining of the portal vein branches was normal. The few septa present contained as a rule proliferated bile ductules, but limited if any inflammatory reaction (Fig. 5).

The hepatic surface, the Glisson's capsule, was focally thickened by accumulation of dense collagenous bundles arranged in an either ellipsoid or nodular fashion. The serosal surface cells were not altered. This nodular accumulation of connective tissue extended into the parenchyma (Fig. 6) and frequently connected with septa extending from the subcapsular portal tracts which were enlarged by fibrosis. In this location some septa were found in every specimen.

Proliferation of sinusoidal lining cells. Throughout most livers, but accentuated in some areas, the sinusoidal lining cells were increased in number. They were particularly conspicuous in areas where the sinusoids were slightly widened (Fig. 7). Some of these cells were macrophages with PAS positive granules, others, also prominent, had an elongated, almost rectangular nucleus which had a loose vesicular chromatin pattern and inconspicuous nucleoli (Fig. 8). Moderate amounts of cytoplasm reacted diffusely in PAS stain, and this reaction persisted after diastase digestion. These cells were tentatively interpreted as activated fibroblasts. A slight excess of perisinusoidal fibers was seen in connective tissue stains in areas where the proliferated sinusoidal lining cells were most numerous. These fibers traversed irregularly the slightly widened perisinusoidal space of Disse which also contained lipocytes.[6] The observations in the paraffin section did not give the impression of an interference with the microcirculation.

Changes of hepatocytes. There were foci consisting of enlarged hepatocytes. The cells had an increased amount of cytoplasm and had nuclei which were large and hyperchromatic. These cells were intermixed with hepatocytes normal in size or even smaller. This hepatocellular change created focal, indistinct nodular areas (Fig. 10). Sinusoidal cell activation was most marked in the areas of hepatocyte enlargement and they also exhibited a distinct sinusoidal dilatation (Fig. 11). These areas had no relationship to lobular topography and were not

sharply limited. The hepatocytes showed no other morphologic evidence of damage and focal necrosis was not more frequent than expected in almost normal specimens. In some of the surgical biopsy specimens, aggregation of segmented leukocytes was noted in sinusoids and sometimes they replaced necrotic hepatocytes. This feature is considered a result of the operation and has no functional or diagnostic implication.[7]

Cholestasis. In two of the specimens focal, chiefly centrolobular, canalicular cholestasis was seen which was accentuated in the centrolobular zone (Fig. 12). The hepatocytes around the bile plugs were not altered and showed little if any bile pigment deposition. The amount of centrolobular lipofuscin in the hepatocytes was normal for the age of the patients. In a few places ductular cholestasis was also observed. The bile ducts were mostly normal but in three cases septal bile ducts were adjacent to a dense infiltrate, mainly of small lymphocytes (Fig. 13). The epithelial cells of these bile ducts had a pale cytoplasm and irregularly arranged nuclei resulting from piling up of the cells which assumed a high columnar shape and surrounded 'stift' cells with dense elongated nuclei.

Liver in Cases with Angiosarcoma

In the instances in which hepatic parenchyma was studied in patients who had angiosarcoma or subsequently developed it, the same alterations as just described were found; sometimes the changes were even more conspicuous. For instance, the hepatocellular activation

had proceeded to the formation of nodules with two or more cell thick plates converging toward the center of the nodule (Fig. 14). In two instances, the septa linked portal and central canals which in one of the cases resulted in a picture of cirrhosis. Of major interest was the frequency of the focal, conspicuous dilatation of sinusoids which was not related to lobular topography. The sinusoidal dilatation was not the result of passive congestion, particularly since the hepatocytes in such areas were not compressed but were rather hyperplastic and free of iron or lipofuscin pigment (Fig. 15). The areas of dilatation were far more numerous than in the patients without angiosarcoma and the sinusoidal lumen was focally wider than in the two biopsy specimens taken before development of angiosarcoma. These areas were multiple and there the sinusoidal cells were more numerous and aggregated, particularly those with diffuse PAS-positive cytoplasm, and the perisinusoidal fibrous tissue also appeared augmented. The sinusoidal lining cells piled up and in four instances this was associated with nuclear and cytoplasmic changes. These hyperchromatic nuclei were larger, and sometimes were huge and irregularly shaped, and the PAS reaction of the cytoplasm was absent (Fig. 16). Small clusters of polymorphous cells offered suggestive microscopic evidence of early focal angiosarcomatous transformation.

Splenic Alterations

In 5 cases information and specimens of the spleen were available for review (Table 1). The enlarged organs (from 560 to 1050 gm) of cases with and without hepatic angiosarcoma all showed grossly visible and conspicuously enlarged Malpighian follicles separated by a meaty-appearing homogenous red pulp (Fig. 17). Microscopically, the follicles contained large germinal centers exhibiting phagocytosis. The periarterial lymphatic sheath was also markedly enlarged, contributing to the unusual size of the follicles, and there was some merging of adjacent follicles (Fig. 18). Sometimes, recent hemorrhage was noted around the penicillary arteries and in the perifollicular zone. In one case there were typical Gamna-Gandy bodies (Fig. 19). The sinuses in the red pulp were dilated and lined by enlarged cells in variable arrangements. In places they lined up with elongated cuboidal shape to produce a gland-like appearance but without evidence of phagocytosis (Fig. 20). This pseudoglandular arrangement was even more impressive when the connective tissue fibers were stained (Fig. 21). The pulp cords were conspicuously thickened and infiltrated by many lymphoid cells of various types and a large number of erythrocytes but without conspicuous iron accumulation.

Arsenical-induced fibrosis

The cases with inorganic arsenical-induced fibrosis had changes similar to those described under the fibrotic precursor stage after VC exposure. Again, portal fibrosis varied in degree throughout the livers and interruption of the limiting plates by fibrosis and connective tissue encroachment on the portal vein branches was seen. Moreover, activation of sinusoidal lining cells with focal borderline widening of sinusoids was accompanied by the characteristic focal activation of hepatocytes.

DISCUSSION

The morphologic investigation of the livers of the workers engaged in the polymerization of VC is to date only preliminary since it was performed so far without adequate clinical/pathological correlation and without ultrastructural analysis. Nevertheless, the study suggests an evolution which assumes an effect of either VC or of its metabolites, presumably formed in the liver, on both hepatocytes and sinusoidal lining cells. This postulated evolution will have to be confirmed in animal experimental studies, particularly since it has been established that inhalation of VC produces in rats and mice angiosarcoma of the liver as well as tumors in other areas[8,9,10] as is being reported at this conference. The effect of the active agent is noticeable on both hepatocytes and sinusoidal lining cells

of the liver and presumably also on cells of the spleen. In the liver, there is apparently an interaction between the stimulated epithelial cells and the mesenchymal cells. The activation of the sinusoidal lining cells is accompanied by fibrosis in portal tracts, within the lobules and particularly in the Glisson's capsule. The focal subcapsular fibrosis is the single most conspicuous lesion in the precursor stage. It is visible on gross inspection during surgery and has been most elegantly demonstrated by peritoneoscopy by Lelbach at this meeting.[2] It is contiguous with the portal fibrosis. Just as the subcapsular fibrosis is focally accentuated, the portal fibrosis varies greatly in extent and appearance. These variations progress in places to a most impressive lesion, particularly where it is localized around proliferated bile ductules. As is well known for congenital hepatic fibrosis, inflammation of such areas may set in resulting in a focal pericholangitis[5] which might account for the irregularly distributed cholestasis found in some cases and also observed in the German material.[2]

The portal fibrosis in the VC-PVC workers is not pathognomonic because similar or identical fibrosis occurs in other circumstances. The fibrosis appears to be progressive, particularly where it encroaches upon the peripheral part of the hepatic parenchyma and where it disrupts the continuity of the limiting plate. A major question is whether the portal fibrosis causes the portal hypertension, present in

some cases either by compression of the portal vein branches or by extension into the walls of the portal vein branches. Such effects have been postulated in the cases of 'idiopathic portal hypertension' reported from India[11] but it is hard to substantiate. In our cases intimal alterations, as described in hepato-portal sclerosis,[12] were not found. The functional significance of the portal fibrosis may become obvious in the rare instance where it leads to septa formation and progresses to cirrhosis, although the latter was only observed in the presence of angiosarcoma.

The slight, focal, intralobular fibrosis is also not pathognomonic because it is frequently encountered in elderly patients and especially in patients with diabetes mellitus. Perisinusoidal fibrosis is associated with an activation of the sinusoidal lining cells and the impression is gained that these are fibroblastic cells. However, light microscopic identification of these cells, which have diffuse non-glycogenic PAS reaction of the cytoplasm, as fibroblasts is only suggested. Fibroblasts in the hepatic parenchyma are supposedly transformed lipocytes.[6] These cells, originally described by Ito,[13] are sinusoidal cells which are devoid of phagocytic activity and are characterized by small fat droplets. These droplets contain much vitamin A demonstrated by fluorescence microscopy.[14] These cells have been designated as the resting form or precursor of fibroblasts.[15,16] The assumption can thus be made that the active agent following VC exposure stimulates transformation of these cells into fibroblasts.

These considerations are supported by the observation that in hypervitaminosis A abundance of the lipocytes is associated with intralobular fibrosis, also accompanied by portal hypertension.[17] Similar cells have been found by both fluorescence microscopic examinations for vitamin A[18] and ultrastructurally in the normal lung. Alterations of pulmonary function have been reported in the VC-PVC workers[19] suggesting a similar activation of mesenchymal cells in the lung.

The activation of the sinusoidal lining cells becomes more significant when it is associated with focal sinusoidal dilatation. The irregular distribution of the widened sinusoids throughout the lobular parenchyma cannot be explained by hemodynamic factors and is associated not only with perisinusoidal fibrosis, but also with a transformation of the lining cells to what appears to be the early stage of angiosarcoma. This is histologically characterized by dedifferentiation of the sinusoidal lining cells the cytoplasm of which loses its characteristic diffuse PAS reaction. These dedifferentiated, presumably multipotential, cells assume then only tectorial function to envelop hepatocytes. This represent the initial step in malignant transformation leading to vessel formation and also fibroplasia by either tumorous or accompanying fibroblasts.

The hepatocytes in these locations are distinctly hypertrophic as well as hyperplastic. The cause of the activation of the hepatocytes may be either a primary stimulation by VC or a response to a hepatocellular injury caused by the reagent. The literature records observations on VC induced hepatocytic injury[20,21] which are, however, not easily

understood because of uncertainities in nomenclature. The combination of hepatocellular hyperplasia with sinusoidal dilatation and sarcomatous transformation of the lining cells is characteristic of the early VC induced angiosarcoma. This gives particular significance to the focal activation of the hepatocytes which may have also functional importance. Whether it progresses to carcinoma after VC exposure is so far only suggested by cases with less clear relation to VC exposure than in the VC polymerization workers.

The alterations of the spleen in the presence of fibrotic hepatic lesions which are not conspicuous and extensive enough to explain hemodynamically portal hypertension by interference with hepatic microcirculation requires special consideration. The histologic changes in the spleen, particularly the enlargement of the follicles and the proliferation of the red pulp cells, suggest that these lesions are not the result of the portal hypertension but rather of a primary stimulation of the splenic lymphoid and reticuloendothelial cells. This is supported not only by the enlargement of the lymph follicles which in portal hypertension secondary to cirrhosis are small, but also by the absence of splenic fibrosis, which is characteristic for mechanically induced portal hypertension.[22] The severity of portal hypertension in our patients is reflected in the periarterial hemorrhages in the spleen and the subsequent formation of Gamna-Gandy bodies. Thus VC exposure sets in motion a process starting with activation of mesenchymal cells and hepatocytes and results in three main manifestations; namely, hepatic fibrosis, splenomegaly and

angiosarcoma. The development of cirrhosis and primary hepatic carcinoma is so far only suspected (Table 2).

Chronic exposure to another chemical agent, namely inorganic arsenicals, results in a similar evolution. 'Idiopathic portal hypertension' with inconspicuous hepatic fibrosis has recently been reported after prolonged treatment with Fowler's solution for psoriasis.[4,23,24] Following long-time treatment, moreover, hepatic angiosarcoma has been reported at least once[25] and even more fascinating are the older observations on workers in European vineyards who were exposed to pesticides containing inorganic arsenicals.[26,27,28] These workers developed hepatic fibrosis, possibly cirrhosis, as well as angiosarcomas and apparently far less frequently, primary hepatic carcinoma. The occurrence of the angiosarcomas was the reason for the discontinuation of the use of these pesticides in vineyards. Examination of the livers of persons with arsenical induced portal hypertension revealed the same changes in the fibrotic precursor stage as after VC exposure. Activation of hepatocytes was also found. In the published descriptions of the arsenical angiosarcomas, the dilatation of hepatic sinusoids and the peculiar envelopment of hyperplastic hepatocytes are also observed.[25,26] It appears that the combined activation of hepatocytes and sinusoidal lining cells of the liver as well as of the splenic cells results from both types of exposures. In arsenical intoxication cholestasis has been reported

as well as what appears to be cirrhosis associated with a chronic active hepatitis.[29] Another apparently rare association with hepatic fibrosis is the reported angiosarcoma in hemochromatosis.[30]

The combination of inconspicuous hepatic fibrosis with severe splenomegaly and portal hypertension is an old puzzle in medicine symbolized by the term "Banti's syndrome" as distinguished from cirrhotic splenomegaly.[31] The key problem in Banti's syndrome is the cause of the portal hypertension which seemingly cannot be explained by the hepatic fibrosis and therefore the lesion is frequently designated as 'idiopathic portal hypertension'.[12,32,33] It has been claimed, however, that the splenomegaly is the result of the inconspicuous hepatic fibrosis or alternatively that the hepatic fibrosis is the result of the portal hypertension, particularly if it is associated with alterations of the portal vein tributaries (hepato/portal sclerosis[12]) or even that the same irritant acts simultaneously on hepatic and splenic cells in the sense of the old concept of hepato/splenic diseases of Eppinger.

The pathogenesis of the splenomegaly in cirrhosis is not always clear. With the development of the concept of portal hypertension by McMichael[34] and others[35,36,37] much of the emphasis in the explanation of splenomegaly has been on a mechanical or congestive basis. Splenomegaly, however, even in cirrhosis does not run parallel with the degree of portal hypertension. Therefore splenomegaly in cirrhosis

is not entirely explained by mechanical factors but also by a direct effect on the splenic tissue, either by the etiologic agent of the liver disease or by liver cell breakdown products.[38] Other observations suggested an immunologic factor. Hypergammaglobulinemia in chronic liver diseases is mainly explained by a lack of sequestration of enterogenous and other antigens by the Kupffer cells either from faulty function of these cells or in fully developed cirrhosis by the bypass of blood from the hepatic parenchyma.[39,40] This creates access of antigens to lymphoid tissue. Thus, even in cirrhosis, in addition to the mechanical component, a primary stimulation of the splenic cells is partly responsible for the splenomegaly. Many years ago, it was demonstrated[41] that in ethionine intoxicated rats the spleen was six times its normal size during the stage of acute hepatic fibrosis but was reduced to twice its normal size in the scar stage when acute liver injury had ceased. These studies thus separate the contribution of mechanical from irritating factors causing splenomegaly.

In VC induced alterations, and probably also in chronic arsenical intoxications, one might accept the third above mentioned explanation; namely, a simultaneous stimulation of both hepatic and splenic cells. The latter probably accounts for the splenomegaly. Its variable incidence probably depends on the degree of splenic cell irritation. The portal hypertension might be explained by the increase in blood flow to the enlarged spleen which has to be

accommodated in the intrahepatic portal venous system and the sinusoidal bed. The inconspicuous portal tract fibrosis might inhibit portal vein distension but even greater may be the effect of the impressive subcapsular fibrosis preventing distension of the sinusoids. Thus even an inconspicuous hepatic fibrosis may cause either presinusoidal or sinusoidal portal hypertension in the presence of splenomegaly. In one patient, indeed, slightly elevated hepatic wedge pressure has been observed by Berk as reported at this conference. The splenomegaly might account for hematologic manifestations, particularly thrombocytopenia as observed in Germany.[2] The accumulation of erythrocytes in the pulp cords of the spleen suggests entrapment and hemo sis.

These studies support one explanation of the Banti's syndrome and are in keeping with experimental observations in which by injection of foreign proteins such as egg albumin[42] and other proteins[43] or of estrogen with cholesterol[44] the characteristic Banti manifestations in the spleen were produced associated with fibrotic changes in the liver. Banti's syndrome is found sporadically in the Western World.[12,32] It appears to be far more frequent in India,[45,46], Uganda[47] and apparently also in Japan[48] where extensive studies on this problem are being done. Today, the etiology in all these instances is not established. The experiences with VC exposure, and also with that to inorganic arsenicals, suggest a search for toxic, possibly environmental, factors in instances of Banti's syndrome of unknown etiology.

The frequency of the described precursor hepatic lesion in relation to angiosarcoma is difficult to establish. In our own material angiosarcomas are more frequent while in the reported German, Rumanian, and Russian studies the non-angiosarcomatous hepatic lesions seem more common. The detection of the precursor lesion is probably an indication to remove the worker from further exposure. However, it is not clear whether the process can be arrested despite the long exposure required to produce the lesion, particularly in animal experiments.

SUMMARY

Preliminary review of hepatic biopsy and autopsy specimens obtained from workers engaged for prolonged periods in the polymerization of vinyl chloride indicates a fibrotic precursor lesion in the liver. It is the only lesion in some instances but also was found in the uninvolved liver of patients with angiosarcoma and in two instances in liver biopsy specimens obtained before angiosarcoma developed. This precursor stage is characterized by a conspicuous subcapsular fibrosis, a non-pathognomonic, progressive portal fibrosis, and a borderline increase of intralobular connective tissue, all associated with focal stimulation of sinusoidal lining cells and hepatocytes. This precursor stage is often accompanied by splenomegaly with enlarged Malpighian follicles and in some instances by portal hypertension requiring portacaval shunt because of variceal hemorrhage.

The portal hypertension is explained by increased splenic blood flow in the face of non-distensible fibrotic portal tracts as well as Glisson's capsule. Transition to angiosarcoma is preceded by focal dilatation of sinusoids with even greater activation but dedifferentiation of their lining cells. This lesion is presumably the result of stimulation of various hepatic as well as splenic cells by vinyl chloride or its metabolites. The demonstrated evolution is identical with that following prolonged exposure to inorganic arsenicals. It is postulated that also other instances of inconspicuous hepatic fibrosis associated with splenomegaly and often with portal hypertension, a combination designated as Banti's syndrome or 'idiopathic portal hypertension', are the result of a toxic, possibly environmental agent, particularly since the disease is found with greater frequency in some parts of the world. The delineation of the fibrotic precursor stage in the liver may assist in the epidemiologic studies of the vinyl chloride induced injury.

LIVER & SPLEEN ALTERATIONS IN VINYL CHLORIDE-POLYVINYL CHLORIDE WORKERS

	FIBROTIC PRECURSOR STAGE 5 CASES 6 SPECIMENS	WITH ANGIOSARCOMA 6 CASES 12 SPECIMENS
FIBROSIS	5/6	
SUBCAPSULAR	5/6	6/9
PORTAL	6/6	12/12
PERISINUSOIDAL	6/6	4/4
FIBROBLASTIC ACTIVATION	6/6	11/12
SEPTA FORMATION	3/6	11/12
SINUSOIDAL DILATATION	2/6	10/12
MICROSCOPIC ANGIOSARCOMA	0	4/12
HEPATOCELLULAR ACTIVATION	5/6	11/12
CHOLESTASIS	2/6	8/12
PERICHOLANGITIS	3/6	5/12
SPLENIC CHANGES	3/3	2/2

Table I

POSTULATED EVOLUTION OF HEPATIC AND SPLENIC LESIONS AFTER VINYL CHLORIDE EXPOSURE

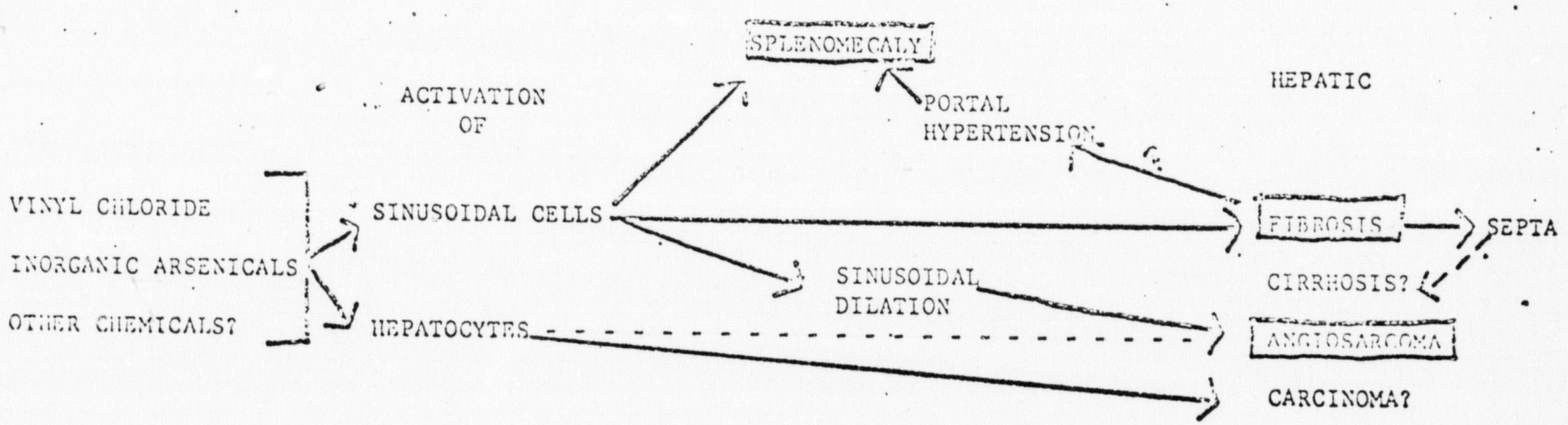

Table II

LEGENDS

Fig. 1. Biopsy specimen. Low power view of fibrotic precursor stage. Enlargement of portal tracts varying in degree throughout the liver, most conspicuous below the capsule. The portal tracts are mostly round and the lobular architecture is intact. (Aniline blue, 5x)

Fig. 2. Biopsy specimen. Fibrosis of portal tract with connective tissue fibers separating periportal part of liver cell plates to render limiting plates indistinct. Loose inflammatory infiltration around proliferated bile ductules (H + E, 210x)

Fig. 3. Biopsy specimen. Impressive fibrosis of excessively enlarged portal tract particularly around proliferated and dilated bile ductules. Inflammatory exudate particularly near the border to the parenchyma where the limiting plate appears destroyed by both fibrosis and cellular exudate. (H + E, 100x)

Fig. 4. Biopsy specimen. Fibrosed portal tract. Collagen bundles separating smooth muscle layers in the wall of the portal vein branch. Some inflammatory exudate and indistinct border towards the parenchyma is noted. Septum-like extension of the portal tract in right upper corner contains proliferating bile ductules . (H + E, 100x)

Fig. 5. Biopsy specimen. Wavy connective tissue membranes extend from the portal tract into the parenchyma. Hepatocytes vary in size. In places the membranes are approximating to form septa. (Aniline blue, 130x)

Fig. 6. Biopsy specimen. Nodular subcapsular fibrosis produces an uneven appearance of surface; some connections of Glisson's capsule with enlarged portal tracts are seen. (H + E, 250x)

Fig. 7. Biopsy specimen. Increase in number and size of sinusoidal lining cells. Many have ellipsoid nuclei and diffusely PAS positive cytoplasm and project pillow-like into the often dilated sinusoids (arrows). (PAS, 560x)

Fig. 8. Biopsy specimen. Lining cells project into lumen of sinusoids. Some have diffuse PAS positive cytoplasm (straight arrow); some granular cytoplasm (curved arrow). (PAS, 1000x)

Fig. 9. Biopsy specimen. Focal increase of perisinusoidal connective tissue fibers. Wavy membranes traverse the tissue space of Disse (Aniline blue, 480x)

Fig. 10. Biopsy specimen. Large hepatocytes with large, hyperchromatic and sometimes double nuclei are intermixed with a few normal sized hepatocytes. The focus of enlarged hepatocytes is not sharply limited and the sinusoids are not dilated. (H + E, 110x)

Fig. 11. Biopsy specimen. Focal sinusoidal dilatation within the lobule around large hepatocytes some of which are binuclear (arrow). The sinusoidal lining cells are prominent and bulging. They pile up in places (curved arrows). (H + E, 225x)

Fig. 12. Biopsy specimen. Bile plugs in dilated canaliculi (arrows) in area with proliferated sinusoidal lining cells. (H + E, 400x)

Fig. 13. Biopsy specimen. Dense accumulation of mononuclear cells near septal bile duct encroaches upon its wall. The bile duct lining cells are piled up and have a pale cytoplasm. (H + E, 130x)

Fig. 14. Autopsy specimen. Regenerative nodule consists of two or more thick cell plates in arrangement different from that of the lobule. (H + E, 60x)

Fig. 15. Biopsy specimen. Focal, conspicuous sinusoidal dilatation with prominence of lining cells which form in places nodular accumulations (arrow). Subsequently angiosarcoma was detected in the patient. (H + E, 150x)

Fig. 16. Close-up of Fig. 15. Proliferation of sinusoidal lining cells many of which have hyperchromatic nuclei and PAS negative cytoplasm. (PAS, 400x)

Fig. 17. Cross section of spleen weighing 1050 gm; surgically removed. The Malpighian follicles are prominent and the red pulp is meaty.

Fig. 18. Large Malpighian follicles in spleen. Their germinative centers are large. Their perifollicular zones merge. The sinuses are dilated and the pulp cords are thickened and cellular. (H + E, 50x)

Fig. 19. Gamna-Gandy body characterized by calcium deposition. Hemorrhage (arrow) near large follicle in spleen. (H + E, 40x)

Fig. 20. Thickened splenic pulp cords contain many mononuclear cells and red cells (curved arrow). In places (arrow) the lining cells of the sinuses form a continuous layer. (H + E, 400x)

Fig. 21. The pulp cords reveal a barely increased fibrous network which accentuates the gland-like arrangement of the sinus lining cells. (PAS, 400x)

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LIVER DISEASE AMONG POLYVINYL CHLORIDE PRODUCTION WORKERS

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Laszlo Makk, M.D.

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ABSTRACT

A protocol for systematic testing of all employees of a chemical plant is presented. This factory manufactures polyvinyl chloride compounds and resins, Absan compounds and resins, and synthetic rubber. The results have been reviewed, leading to the discovery of two additional cases of angiosarcoma and 11 cases of portal fibrosis. Two of the 11 cases were found to have developed in other than polyvinyl chloride production employees.

The B.F.Goodrich chemical plant in Louisville, Kentucky, manufactures polyvinyl chloride resins and compounds, ABS resins and compounds, and specialty synthetic rubber. Although there are over 200 chemicals used in these manufacturing processes, the principal chemicals used in the production of the polyvinyl chloride are the monomers vinyl chloride, vinyl acetate, and vinylidene chloride, whereas those used for the ABS and rubber production are butadiene, styrene, and acrylonitrile.

These processes are housed in different parts of the factory complex, thus making cross exposure very slight or nonexistent. However, work histories of all employees show that many of the rubber and ABS people have been involved in or worked in the polyvinyl chloride production area at some time or other. Also, the areas of production, other than the polyvinyl chloride production area, may have some very slight exposure to vinyl chloride as in the general plant environment.

In an attempt to relate liver abnormality to the degree of vinyl chloride exposure, five categories of work areas have been determined: The higher exposure is in the area of (1) polyvinyl chloride production. This is followed in order by (2) other areas of production, including the rubber plant, (3) maintenance personnel for the polyvinyl chloride production, (4) all other maintenance employees, and (5) all other employees - including administration, plant protection, secretarial

In 1973 a series of events resulted in a need to review the death certificates of all former B.F.Goodrich employees[1]. This resulted in the discovery of five deaths due to angiosarcoma of the liver. A review of past illnesses of current workers revealed that three employees had had splenectomy and/or a shunt procedure for hypersplenism or portal hypertension. This resulted in the initiation of a series of tests to determine the extent of liver disease in vinyl chloride workers.

As Figure #1 will show, the testing was started with a standard SMA 12, which included the SGOT, LDH, alkaline phosphatase, total bilirubin, albumin, total protein, cholesterol, uric acid, BUN, glucose, phosphorus, and calcium. The blood was drawn from fasting employees, then transported immediately to a local laboratory, where the testing was performed.

As can be seen, if the SMA-12 was reported as normal, the test was repeated in six months. If only one liver function test (SGOT, LDH, alkaline phosphatase, or total bilirubin) was elevated, the test was repeated in three weeks. Should this elevation persist, a battery of liver function tests was then requested. If two or more tests were elevated on the original examination, the battery was requested without delay. In those cases where the battery showed major abnormality, the special procedures and treatment as indicated were carried out. The battery of tests is listed in Table #1.

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These included (in addition to the repeat SMA-12) the fractionation of LDH, alkaline phosphatase, and total bilirubin - SGPT, gamma GT, alpha feto protein, ICD, protein electrophoresis, and a CBC with platelet count.

X-rays consisted of chest X-ray and liver-spleen scan using Technetium 99M sulfur colloid.

The special procedures consisted of selective hepatic and splenic angiograms, as described by Viamonte etal in 1970, including free hepatic vein pressure studies and wedge hepatic vein pressure studies, and hepatic vein flow studies[2]. A liver biopsy was performed when indicated. The first eight cases requiring biopsies were done at laporatomy. All remaining cases have been done through the internal jugular vein at the time of the hepatic vein catherization, using a method first described by Hanafee in 1967 and more recently by Rosch etal in 1973[3,4].

In referring back to Figure #1, testing now continues in those cases showing improvement. If the no. 1 battery is found to be normal, the SMA-12 tests are repeated in six months. If there is questionable or only mild abnormality, then the chemical portion of the battery is repeated in three weeks.

If the no. 2 battery is normal, then a routine SMA-12 is done in three months. But if the no. 2 battery is found to persist or increase in degree of abnormality, then the special procedures and biopsy are scheduled.

Figure #2 will indicate the percentage of abnormalities found on the original examination of 1183 employees by using the SMA-12 tests.

315 individuals had one abnormal test - 26.6%.

41 individuals had two or more abnormal tests - 3.5%.

75 individuals out of the 315 tested for the second time were found to persist in the abnormality. This represented 6.3% of the plant population, or 24% of the second SMA-12 group.

A total of 116 cases had batteries run, or approximately 10% of the total plant population. Fifty-seven of the 116 cases were found to have normal batteries; seven cases were found to have major abnormality, requiring special procedures.

Fifty-two cases had minor or questionable abnormalities; ten of these 52 later showed persistent or increased degree of abnormality, leading to special procedures examination.

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The scans which were reported on 116 employees were broken down into five groups:

1. Patchy uptake - indicating cirrhosis or fibrosis.
2. Enlarged liver - which is of doubtful value.
3. Enlarged spleen - over 14-cm as described by Siegel[5].
4. Cold spot - indicating tumor.
5. Normal liver-spleen scan.

The arterial and venous studies were reported as:

1. Corkscrewing of vessels - indicating cirrhosis or fibrosis.
2. Tumor blush - indicating tumor. (In angiosarcoma the blush persists for 20 seconds.)
3. Reversed portal flow - indicating portal hypertension with collateral circulation.
4. Portal or splenic vein thrombosis.
5. Level of free hepatic vein pressure.
6. Level of wedge hepatic vein pressure.
7. Corrected sinusoidal pressure (#6 minus #5).
8. Normal arterial and venous studies.

To date we have had 17 cases require special procedures, 16 of whom were biopsied. The nonbiopsied case had no abnormal liver tests but showed a defect on the scan which turned out to be a normal functioning displaced gall bladder.

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Table #2 will indicate the location of workers having abnormal SMA-12 examinations. It is seen that there was a greater percentage of abnormalities seen in the areas of production other than PVC. This, however, was of borderline statistical significance. The maintenance and other workers area also showed about the same degree of abnormality.

Table #3 shows the location of abnormal batteries. Here the statistical significance is valid, with 10% for PVC production as opposed to 4.1% abnormal for other areas of production.

Table #4 shows the percentage of individual abnormal tests of the SMA-12 with regard to location. The alkaline phosphatase was seen to be the most frequently elevated test. This could be due to the number of young workers scattered throughout the plant. Fractionation of the alkaline phosphatase elevations shows that only 54% had elevations of the heated fraction of 25% or over. This implies that liver caused the elevation in only 54% of the elevated cases.

Table #5 shows the percentage of abnormal individual battery tests with regard to location. There is no outstanding single test in this group.

Table #6 shows the results of the scan, the special procedures, and of the liver biopsy in 17 cases. Two of the 17 had angiosarcoma. Both are receiving chemotherapy. These two patients also demonstrated periportal fibrosis on the microscopic study of the liver biopsy.

As can be seen, there were five liver biopsies reported as normal - out of a total of 16 biopsies. Three of these five were anticipated, as neither battery, scan, nor special procedures showed any abnormality. The fourth case had minor liver function abnormalities with a cirrhotic scan. The fifth had an elevated liver function test.

Two of the remaining ten abnormal liver biopsies were found in patients who had worked only in other production areas. Both of these patients showed periportal fibrosis, as did the polyvinyl chloride worker. Periportal fibrosis was the most common diagnosis according to biopsy. Two of the eight remaining had splenic vein thrombosis by special procedures tests, including portal venogram at time of surgery. Both had esophageal varices; both had large spleens. Biopsy of both livers showed mild fibrosis.

The findings have resulted in the removal of 59 employees to areas of no exposure to hepatic toxins.

There may be some significance to the results of the fractionation of LDH. Isolated factor no. 4 elevations were seen in 66% of B.F.Goodrich employees, whereas there was an isolated factor no. 4 elevation in the hospitalized patients (other than B.F.Goodrich employees) of only .02%. The reason for this is unknown.

Another finding was the very high incidence of elevation of CPK found in the black worker (75) of B.F.Goodrich as opposed to the white (12). I know of no reason for this.

Of the seven cases of acroosteolysis tested, only one had an abnormal battery. When asked to stop drinking, his battery returned to normal in three weeks.

In conclusion, a protocol for testing has been presented. The results of these tests have been reviewed. It seems that no one isolated test - be it SMA-12, battery, or special procedures - is adequate for a thorough work-up. It is certain that the SMA-12 or batteries without scan will not predict a cold spot in the liver. It is also true that a scan will not always predict fibrosis. The special procedures seem quite accurate in detecting abnormal pressure studies in the liver, indicating fibrosis or cirrhosis, and may be useful in diagnosing angiosarcoma of the liver (due to the persistent tumor blush).

It further seems that the battery of more sophisticated tests will need be run on some of the normal SMA-12 group to see if the SMA-12 is a reliable tool in discovering liver abnormalities or in screening out normal cases.

These studies will be continued through the working years of the employee and probably even after his retirement. Undoubtedly the protocol will change as more information is gathered and as research shows a better method for detecting disease, whether it be angiosarcoma, fibrosis, or portal vessel thrombosis with splenomegalia.

I'm sure that everyone in this room hopes that manufacturing procedures have been or will be developed to such an degree that these abnormalities will not occur and thus will require no detection.

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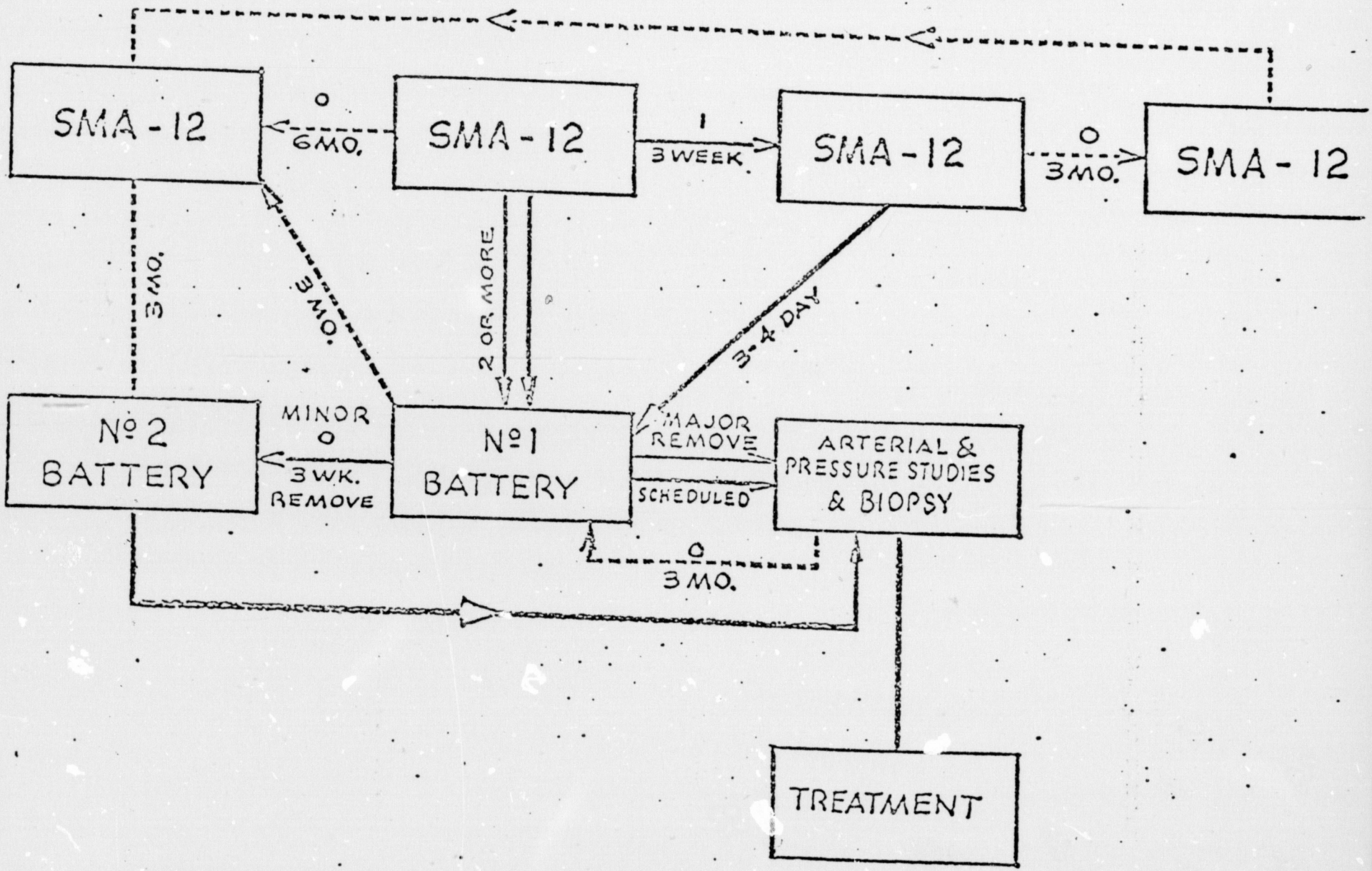
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MINOR
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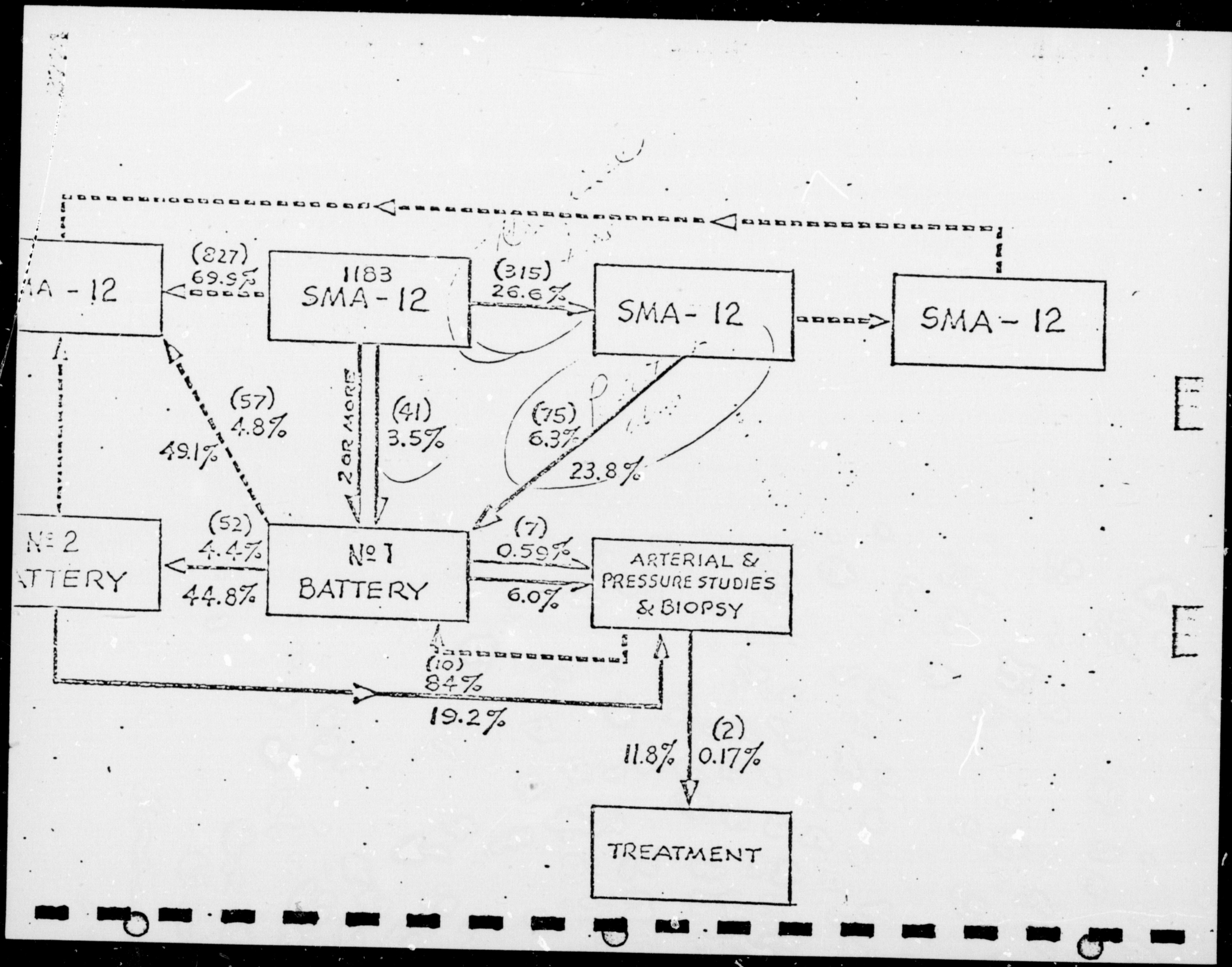
TREATMENT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2 OR MORE
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TREATMENT

NOMENCLATURE

SMA	SEQUENTIAL MULTIPLE ANALYSIS
SMA-12	CALCIUM, TOTAL BILIRUBIN, ALBUMIN, CHOLESTEROL, TOTAL PROTEIN, ALKALINE PHOSPHATASE, LDH, SGOT, GLUCOSE, BU URIC ACID, INORGANIC PHOSPHORUS.
BUN	BLOOD UREA NITROGEN
LDH	LACTATE DEHYDROGENASE
SGOT	SERUM GLUTAMIC OXALACETIC TRANSAMINAS
SGPT	SERUM GLUTAMIC PYRUVIC TRANSAMINASE
GGTP	GAMMA GLUTAMYL TRANSPEPTIDASE
CEA	CARCINO EMBRYONIC ANTIGEN
ICD	ISO CITRATE DEHYDROGENASE
CBC	COMPLETE BLOOD COUNT (WITH PLATELET)
AFP	ALPHA FETO GLOBULIN
FRACTIONATION	PROTEIN ELECTROPHORESIS
	LDH
	ALKALINE PHOSPHATASE
	TOTAL BILIRUBIN
X-RAYS	CHEST "
	LIVER-SPLEEN SCAN

LOCATION OF ABNORMAL SMA-12

AREA	NUMBER EMPLOYEES	NUMBER ELEVATED	% ELEVATED
PVC PRODUCTION	274	59	21.5
OTHER PRODUCTION	411	132	32.2
PVC MAINTENANCE	35	10	28.6
OTHER MAINTENANCE	216	58	26.8
ALL OTHER	247	56	22.6
TOTAL	1183	315	26.6

LOCATION OF ABNORMAL PATTERNS

AREA	NUMBER EMPLOYEES	NUMBER ABNORMAL	PERCENT ABNORMAL
PVC PRODUCTION	274	30	10.9
OTHER PRODUCTION	411	17	4.1
PVC MAINTENANCE	35	0	0
OTHER MAINTENANCE	216	9	4.2
ALL OTHER	247	3	1.2
TOTAL	1183	59	5.0

ABNORMAL SMA-12 BY TEST

AREA	% TOTAL BILIRUBIN	% ALKALINE PHOSPHATASE	% LDH	% SGOT
PVC PRODUCTION	35.6	47.5	3.4	33.9
OTHER PRODUCTION	29.6	50.7	8.3	28.8
PVC MAINTENANCE	10.0	50.0	0	50.0
OTHER MAINTENANCE	17.2	46.5	5.2	51.7
ALL OTHER	33.9	42.8	14.3	41.1

ABNORMAL BATTERY BY TEST

AREA	% SGOT	% GAMMA G.T.	% SGPT	% ALKALINE PHOSPHATASE	% LDH	% TOTAL BILIRUBIN
PVC PRODUCTION	46.9	50.0	34.4	59.4	9.4	43.8
OTHER PRODUCTION	46.2	33.3	20.5	61.5	28.2	41.0
PVC MAINTENANCE	50.0	0	0	50.0	50.0	0
OTHER MAINTENANCE	50.0	44.4	38.9	61.1	16.7	50.0
ALL OTHER	46.7	13.3	6.7	46.7	26.6	40.0

Name	Work Area	Scan	Hepatic Arteriogram	Microscopy
E.P.As.	Maint.	Normal	Normal	Metamorphosis moderate Portal Fibrosis slight
J.L.Bud.	PVC	Large spleen mild Chirrhosis	Aneurysm S.P.T & Hypersplenism	Slight Metamorphosis
J.C.Day	PVC	Chirrhosis	Hepatomegaly	Metamorphosis, Early Cirrhos
K.E.C.A.	PVC	Defect-Chirrhosis	Defect	Fibrosis & Angiocarcoma
C.Hut.	PVC	Large spleen Cirrhosis	Enlarge spleen Aneurysms	Normal liver, sl. Fibrosis Hypersplenism
K.Fisch.	PVC	Possible defect	Displaced G.B.	—
E.Lick.	OP	Cirrhosis	Normal	Slight Portal Fibrosis
R.Reed	PVC	Defect	Defect	Angioscarcoma Fibrosis
L.Mark *	PVC	Normal	Normal	Normal
H.Sellers	PVC	Normal	Normal	Normal
J.S.Stone	OP	Normal	Normal	Portal Fibrosis
Guy K.	PVC	Normal	Normal	Normal
C.L.Wells	OM	Cirrhosis	Normal	Normal
W.H.E.W. *	PVC	Defect or Patch	Cirrhosis with reverse flow	Fibrosis 6 yrs ago
E.Wood **	PVC-6yrs.	Normal	Normal	Hepatitis Cirrhosis
E.Mack	PVC	Normal	Normal	Fatty Metamorphosis
H.Yee	PVC	Normal	Normal	Normal

* Done at patients request.

** Had bad PR. shunt — Non-Alcoholic Cirrhosis.

[redacted] Married 6 yrs. Has been [redacted] elsewhere for past 6 yrs.

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A. General Comments

Management spokesmen during oral presentation and in written submissions (see the ORC statement) continually refer to IUD vs. Hodgson in which case the Court of Appeals attempted to clarify the concept of feasibility. The court failed to do more than manifest the current crystallization of mores.

Essentially unchanged since the beginning of our society, this crystallization is an expression of thinly veneered stiality. We do not accept it. If the history of the labor movement in this country has any meaning, it is that workers should struggle against being forced to eat bad bread.

In the NIOSH presentation, a solution was proffered: the concept of socially acceptable risk. The difficulty with this concept is that it connotes the possibility of mutual agreement but denotes no specific means by which those who are asked to agree can arrive at a rational and humane conclusion.

Only the perverse or the exceptionally naive and ignorant think the determination of consensus should simply be one of subjectively discussing and accepting a given percentage of death and disease at a given so-called feasible limit of exposure.

For example, human lives are at risk for every product. Obviously the social value of the uses of a toxic substance must be considered in calculating a socially acceptable risk. Some uses or products resulting from use should be eliminated. This would reduce the size of the population at risk.

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Other expressions in the calculus should include:

other environmental effects
substitutability of the product, raw material, or process
externalized social costs
desires of the population at risk
genetic and other long-term effects on the human stock
limitation of exposure by mandated administrative controls such as rotation
feasibility of potential technology
future raw material needs
economic and social development

A word or so on economic and social development is pertinent to the tone of these hearings. Economics is a new science which has much to learn from biology.

Remember the dialectic on biological change (development in evolution, embryologically, and even in physiologic adaptation): epigenesis vs. preformation. The historic question was whether growth, for example, proceeded essentially by increase in volume or increase in volume + differentiation (internalized development). The epigeneticists seem to have won. There has been a rejection of Galen's notion, namely, that growth (to use Galen's example) equals the blowing up of a pig's bladder.

Yet the current, simplistic views of "economic" growth are reflected in the pre-occupation with Gross National Product and similar indices of volume.

The economist -- all of us -- must learn that economic development is not the blowing up of a pig's bladder. Without internalization there cannot be that kind of change in the quality of human life identified with satisfactory social development. The concept of social acceptability, it seems to us, must encompass an epigenetic view of human development. It cannot be restricted, for example, to the micro-economics of polymerization of vinyl chloride.

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A regulation that increases the cost of a product, and may thereby reduce the rate at which its market expands (no decrease in actual market of PVC is conceivable, however) also reduces the rate at which a number of other macro-economic effects would occur, such as:

a. problems of disposal

b. depletion of raw materials

c. diversion of capital for control equipment

d. diversion of human resources for planning, construction and operation of controls

e. loss of dollar exchange in importation of the basic petrochemical stock (or its domestic replacement)

f. expansion of health delivery system (preventive and curative) to meet a given level of risk

/Thus, in regard to (f), a no-detectable goal as measured by instrumentation sensitive at 1 ppm bears a risk as high as one death per 100,000 workers. Turnover rate is 5 percent. Thus in 10 years at least one million workers may be exposed with a possible ten cases of angiosarcoma resulting.

The costs of these cases are mentioned below. At 50 ppm 20,000 cases could occur at the same cost per case. Levels of exposure and risk between these extremes would have proportionately calculable costs. (Angiosarcoma is only one of a number of cancers and other pathological manifestations that result from VC exposure./

g. concentration of production among producers least efficient, removing the stimulus for the entry of new firms into the field

From a macro-economic point of view, the SPI proposal encourages the concentration of production in a few firms, externalizes social costs, will result in adverse health effects and proposes a level of control above that which is technically feasible. It does not propose a socially acceptable risk if anyone other than management is to be considered as part of the implied consensus.

B - Further Comments on Biological Effects

Vinyl chloride is a carcinogen. The Departments of Labor and Health, Education and Welfare recognize this fact. But this chemical has other equally adverse effects on human beings: it is a mutagen and a teratogen.

In our plea on May 8 to the Department of Labor for a tighter emergency standard for vinyl chloride we pointed to animal evidence of transplacental effects provided the Secretary in his own hearing on February 15 by a government witness: Dr. Cesare Maltoni. The standard -- we argued -- fails to protect exposed working women. Now we know that even the wives and children of exposed working men are at risk. Vinyl chloride is introduced into the home on the clothes and even on the body of the vinyl chloride worker.

In testimony before the Labor Department on June 25, on their proposal for a permanent VC standard, we noted that one of every four cases of angiosarcoma reported in Sweden from 1958 to 1969 were babies. We pointed to a German study reporting PVC particles ingested and disseminated into the milk of lactating women through the bloodstream (particles expected to contain residual vinyl chloride).

Earlier this month Dr. Irving Selikoff reported data that suggests a higher than normal rate of stillbirths and miscarriages among the wives of VC-exposed workers. The incidence of abnormal chromosomes among workers he examined was higher than expected. While the meaning of this data is being amplified by a crash program at Mount Sinai School of Medicine, Dr. Selikoff has drawn our attention to the work of both Swedish and American investigators which will help us to interpret his data.

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According to Dr. Frederick De Seres, Swedish scientists, using bioassay methods developed in part in his laboratory at the National Institute for Environmental Sciences, will publish (in Ambio) data which conclusively establishes vinyl chloride as a mutagen. In Salmonella-liver microsome studies, they report the rate of mutation from exposure to vinyl chloride to be four times normal. The study has been replicated with approximately the same results by scientists at Stanford Research Institute.

Two weeks ago, in pursuit of our own investigation of the social impact of vinyl chloride and its control, we requested the Ohio State Health Department to determine the rates of fetal mortality and of malformations at birth in cities with substantial numbers of workers involved in vinyl chloride or polyvinyl chloride production. The answer we received from Dr. Peter Infante, the investigating epidemiologist, reports no strong evidence of fetal mortality in the three cities in that state where major production facilities are located.

However, this is not the case for malformations at birth.

During the years 1970 through 1973, the state average for malformations reported at birth in Ohio was 11.4 for each 1,000 live births. In Painesville, where Robintech and Uniroyal have plants, the rate is nearly 20 per 1,000 live births. In Ashtabula, where a General Tire Facility is located, the rate is nearly 18. The plant city for a B. F. Goodrich facility, Avon Lake, experienced the highest rate: nearly 22 reported per 1,000 live births. Dr. Infante reports that these differences, from a statistical point of view, are significantly greater than the state average. However, he is investigating other possible causes unrelated to vinyl chloride that may explain the differences.

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We hope an investigation of the differences exonerates vinyl chloride exposure. Otherwise we face a compelling human disaster that may be unmatched in our time. We conservatively estimate that one and a half million families are or have been exposed to significant quantities of vinyl chloride. (The number of women directly exposed is indicated by the attached analysis.)

Many of the separate lines of evidence discussed here may eventually be explained away. All of it cannot be. These facts, plus the risk of cancer and other diseases, tell us that we are in the midst of a public health episode of immense proportions.

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THE EMPLOYMENT OF WOMEN - FROM THE GENERAL ECONOMY TO VINYL CHLORIDE SPECIFICALLY

All figures are for the year 1972, except where noted. Source: Bureau of Labor Statistics, 1973 annual publication.

1.	Women employees on nonagricultural payrolls	27,223,000
2.	Women employees on manufacturing payrolls	5,353,000
	Durable goods	2,231,000
	Non-durable	3,122,000
3.	Women employees in chemicals and allied products	206,600
	rubber and plastics products	204,000
	Total	410,600
4.	Number of married women, husband present, with children, in the labor force (March 1972)	19,249,000
	With children under six years of age	3,746,000
	With children six to 17 years only	6,706,000
	With children under 18 years	10,452,000
	With none under age 18	8,797,000
5.	Employed, married women, husband present	18,217,000

6. Male-female ratio:

	Manufacturing	%	Chemical and Allied	%	Plastics and Rubber	%
Male workers	13,580,000	72	795,600	79	423,000	67
Female workers	5,353,000	28	206,600	21	204,000	33
Total	18,933,000		1,002,200		627,000	

	Chem-Allied and Rubber-Plastics	%
Male workers	1,218,600	74.8
Female Workers	410,600	25.2
Total	1,629,200	

7.	Estimated VC production, polymerization, compounding and fabrication workforce	700,000
	Possible female worker population (25%)	175,000
8.	Percentage of women (in the workforce) who are married, with husband present, and with children under 18	39%

March 1972 figures: 10,452,000 (those with children under 18)
27,223,000 (women employed generally)

Table -2-

8. (continued)

Using this percentage yields:

Manufacturing	working women, married	2,087,670
Chemical-Allied and Rubber-Plastics	with children under 18	160,134
Vinyl Chloride Workforce		68,250

9. Notations:

A. The percent of employees who are not production workers is not factored in.

Manufacturing - 27% are non-production personnel
Chem-Allied - 42%
Rubber-Plastics - 22%
Chem-Allied and
Rubber-Plastics- 34%

B. The 39% figure of women who are married, with husband present, and children under 18 is a labor force number over an actual employment number, and thus not a strict comparison.

TURNOVER RATES

In considering the worker population exposed to a hazard, the following turnover rates should be factored in:

	Accessions	Separations	
Manufacturing	4.4%	4.2%	(for 1972)
Chemical & Allied	2.2%	2.2%	
Rubber & Plastics	5.1%	4.7%	

(These rates are for employees generally, not for production workers specifically)

Source: Bureau of Labor Statistics, 1973.

From a biological point of view, as even some of the management spokesmen indicated (Gehring of Dow et al) in the course of these hearings, unnecessary exposure should be avoided. These statements were made in light of carcinogenic effects. Teratogenic and mutagenic effects occur at lower levels of exposure over much shorter periods of time. The need for control becomes, then, both more urgent and at a greater level of stringency.

Just as no "safe" level of exposure from a carcinogenic point of view has been or can be demonstrated at this time, from a mutagenic or teratogenic point of view it will be more difficult to establish a dose-effect relationship at measurable levels. Yet, to meet the mandate of the Occupational Safety and Health Act, the standard for vinyl chloride should aim at preventing adverse effects on the most sensitive organic systems of the most sensitive portion of the population at risk.

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C. Feasibility of the Proposed Vinyl Chloride Standard

Ronald J. Ruskan
Sheldon W. Samuels

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